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ZYGOMATIC INFECTIONS AS A FACTOR IN OTITIC COMPLICATIONS

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In modern surgical operations on the mastoid one of the prime considerations of the operator is the complete exenteration of all the cellular structure. It is an accepted fact that the incomplete operation prolongs the morbidity greatly, and also that the incidence of otitic complications is much higher in the incompletely performed operations than in the complete ones. By complete operation we mean the type first popularized by Schwartze[1] and later modified by Gruening,[2] Whiting[3] and Friesner.[4] At the end of the complete operation the surgeon should be able to see clearly the external semicircular canal, a clean initial groove, a clean sinus plate, the middle fossa plate, the digastric muscle, the mastoid tip removed and a cleanly exenterated zygoma. In this communication we are concerned chiefly with the consideration of the zygoma.

ANATOMY

The zygomatic (old term, malar) bone forms the prominent part of the cheek; it is located lateral to and below the orbit, and it forms the lateral border of the orbital cavity. It rests on and is united to the maxilla below. Behind, it enters into the formation of the zygomatic arch. The zygomatic bone has three surfaces, namely, the malar, the orbital and the temporal; and two processes, the frontosphenoidal and the temporal. The temporal process projects backward and articulates

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From the otologic service of Dr. I. Friesner, Mount Sinai Hospital.

1. Schwartze, Hermann: Lehrbuch der chirurgischen Krankheiten des Ohres, Stuttgart, Ferdinand Enke, 1885, p. 338.

2. Gruening, E.: Notes on Operations upon the Mastoid Process, New York M. J. **55**:11, 1892; The Mastoid Operation in Acute Empyema and in Caries, Mt. Sinai Hosp. Rep. **1**:281, 1899; Casuistisches Beiträge zur Eröffnung des Processes mastoideus bei acuter Otitis media purulenta, mit Betheiligung der Warzensellen, Munich, J. F. Bergmann, 1879; Ztschr. f. Ohrenh. **8**:297, 1879.

3. Whiting, F.: The Modern Mastoid Operation, Philadelphia, P. Blakiston's Son & Co., 1905.

4. Friesner, I., quoted by Maybaum, J. L.: Minutiae of Surgical Technique of Simple Mastoidectomy, Laryngoscope **35**:265 (April) 1925.

by an oblique serrated extremity with the zygomatic process of the temporal bone. It is in the latter structure that we, as otologists, are greatly interested.

The zygomatic process of the temporal bone springs from the anterior and lower part of the temporal bone by two roots and curves forward horizontally, where it ends in a serrated edge which articulates with the temporal process of the zygomatic bone. The upper border of the bone is continuous with the posterior root. The posterior root is a salient ridge that extends backward above the external acoustic meatus and is continuous with the linea temporalis of the parietal bone. The lower border of the zygomatic process is continuous with the anterior root, which is a broad, thick bar of bone that turns abruptly in a medial direction to join the side of the skull. The lateral end of the root is prominent and is called the tubercle of the root of the zygoma. The medial end is continuous with a smooth transverse elevation of the lower surface of the squama, called the articular tubercle. This forms the boundary of the mandibular fossa.

EMBRYOLOGY

The temporal bone is ossified from eight centers exclusive of those for the internal ear and the tympanic ossicles. We are interested in the ossification centers for the squama, including the zygomatic process. The squama is ossified in the membranous bone from a single nucleus which appears near the root of the zygomatic process about the second month. The squama unites with the tympanic ring shortly after birth. The petromastoid part unites with the squama about the first year. The subsequent changes in the squama and zygoma are in conjunction with the development of air cells in the mastoid process and with the change in the mandibular fossa, which is at first very shallow and looks lateralward. The fossa deepens and looks downward; the change in direction occurs as follows: The part of the squama which forms the fossa lies at first below the level of the zygomatic process. As the base of the skull increases in width, this lower part of the squama is directed horizontally inward to contribute to the middle fossa of the skull, and its surfaces come to face upward and downward. The attached portion of the zygomatic process also becomes everted and projects like a shelf at right angles to the squama.

The size and shape of the zygoma vary in different persons and in different races (Martin [5]). Dolichocephalic skulls frequently have large pneumatized zygomas.

Just as the histologic structure of the mastoid varies, so too does it vary in the zygoma. As in the mastoid, the cell structure in the zygoma may be of the pneumatic (fig. 1), diploetic, sclerotic (fig. 2) or mixed

5. Martin, R.: Lehrbuch der Anthropologie, Jena, Gustav Fischer, 1914.

type (Zuckerkandl [6] and Bélinoff and Balan [7]). The pneumatic mastoid is frequently associated with a pneumatic zygoma, but it is not unusual to note the occurrence of a diploetic or a sclerotic zygoma with a pneumatized mastoid bone. The mixed cell type of zygoma, as in the mastoid, is the most frequent (Zuckerkandl). Faulty pneumatization in the development of the zygoma in all probability takes place as in the mastoid, according to the theory of Wittmaack.[8]

PATHOLOGY

The vast majority of zygomatic infections are secondary to involvement of the mastoid. It is granted that, all other considerations being the same, infections will spread more rapidly through a pneumatized zygoma than through a nonpneumatized one. As the infection

Fig. 1.—X-ray picture of a large pneumatic cell mastoid with well pneumatized zygoma (*a*).

progresses it may perforate the cortex anywhere in its course. The cortex is less dense at the base of the zygoma, particularly the inner table, than it is elsewhere, and consequently most perforations take place there. As a result, an extradural abscess of the middle fossa is not an infrequent complication of zygomatic disease. If perforation through the cortex of the anterior or posterior root occurs, an abscess of the soft part may result. Swelling and inflammation of the soft part may accompany an early periostitis.

6. Zuckerkandl, E.: Makroskopische Anatomie, in Schwartze, Hermann: Handbuch der Ohrenheilkunde, Leipzig, F. C. W. Vogel, 1895.

7. Bélinoff and Balan: Ueber der Bau der Pyramidenspitze, Monatschr. f. Ohrenh. **64**:1185, 1931.

8. Wittmaack, K.: Zur Frage der Bedeutung der Mittelohrentzündungen des frühesten Kindesalters für später, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **129**:207 (July 21) 1931.

SYMPTOMS AND DIAGNOSIS

Swelling over the side of the face associated with pain and tenderness is the most frequent symptom. It is of significance that this symptom may appear with the cessation of the aural discharge. This is particularly true in children. This symptom may occur prior to the original mastoidectomy, or may follow it. It is true that infection of the zygoma may exist without any demonstrable symptoms. Roentgenologic examination is of inestimable value in establishing a diagnosis. It is not uncommon to note that patients complain of pain in the eye. This may often lead to the erroneous assumption that the patient might have an osteitis of the petrous pyramid, especially if the complaint is associated with a profuse postauricular or middle ear discharge after mastoidectomy. We have seen just such a case in which the entire

Fig. 2.—X-ray picture, showing marked periantral sclerosis with some cellular structure in the zygoma (*c*).

symptomatology disappeared after exenteration of a carious focus in the zygoma and evacuation of an epidural abscess in the middle fossa. There are no symptoms which are distinctive of zygomatic disease.

REPORT OF CASES

CASE 1.—B. L., aged 8, was operated on at a private sanatorium two years before admission to the hospital. Despite all treatment the patient had a persistently draining, postauricular fistula. On admission to the otologic service of Dr. Friesner, it was noted locally that just above the center of a firm postauricular scar corresponding to the situation of the antrum a fistulous opening presented, which extended forward, inward and upward. The wound was reopened. A huge cellular zygoma was found from which frank thick green pus exuded. The zygoma was carefully exenterated, the fistulous tract was excised, and the wound was closed. In two weeks the patient was discharged, healed.

Comment.—This case is illustrative of the only too common lack of precaution on the part of the operator in investigating the posterior root of the zygoma. Frequently in acute mastoiditis one encounters several good-sized cells just external to and in front of the antrum. These zygomatic cells are filled with pus and granulations, especially when suppurative mastoiditis has existed for any length of time. When one fails to clean out this carious focus of the bone, one leaves a contributing cause for malunion, fistula or possibly an intracranial complication.

CASE 2.—B. S., aged 14, with a three weeks' history of pain in the right ear, was admitted to the otologic service of Dr. Friesner. A myringotomy was performed. Discharge was only moderate in amount, and lasted for two weeks. One week before admission a nonmovable, tender swelling developed over the middle portion of the zygoma, and there was a cessation of the aural discharge. Mastoidectomy was performed on the right side. The mastoid cells and the cells of the zygoma were filled with mushy granulations, but no free pus was encountered. Culture of the secretion in the zygoma was negative. The exenteration of the zygoma was carried forward about 1½ inches (3.77 cm.), but no site of perforation could be located grossly. The postoperative course was uneventful; the swelling over the zygoma eventually disappeared. The patient made a complete recovery.

Comment.—The history of pain in the ear for three weeks, with the suggestive cessation of an aural discharge associated with the sudden swelling over the zygoma, viewed in conjunction with a cloudy x-ray picture especially over the zygoma, led us to immediate exploration. From our findings we felt that we were dealing with a minute perforation of the cortex of the zygoma with marked periostitis.

CASE 3.—B. S., aged 6, was admitted to the hospital with frank evidence of acute mastoiditis. A simple mastoidectomy was performed. The mastoid and zygoma were extensively diseased. It was noted at the original operation that the patient had a large cellular zygoma, and that the posterior root had been cleaned out apparently to the limit. Culture of the pus was reported as showing Streptococcus haemolyticus (beta). The patient did not do well, but continued to have a low grade fever ranging from 99 to 102 F. There was also a moderate discharge from the middle ear and the mastoid. On the tenth day after operation, a tender swelling became evident over the zygoma about three-fourths inch (1.87 cm.) from the ear. This became soft and fluctuated and was adherent to the zygoma. It was aspirated, and pus was obtained. Incision and drainage were instituted. Culture of the aspirated pus was again reported as showing Streptococcus haemolyticus (beta). During the exploration bare bone could be felt. Ten days later the wound had entirely healed, and the patient made a complete recovery.

Comment.—In cases of this type, in which one apparently reaches the end of cellular structure in the zygoma, and despite this a subperiosteal abscess with perforation into the infratemporal fossa or the soft parts occurs, we can postulate several explanations. First, there may be a microscopic communication with cells further anteriorly,

despite the fact that grossly the zygoma is apparently free of cells. Secondly, there may be extension of the disease process along the cortex of the zygoma via the blood vessels. Finally, the focus may be laid down prior to the operation and involve the cortex and periosteum; then in spite of the removal of the cellular structure of the zygoma, the disease process may proceed to perforation. One must bear in mind, also, that any combination of these factors may exist in the same case.

Case 4.—S. K., aged 20, first entered the medical service of Mount Sinai Hospital in 1926, at the age of 14, with diabetes of moderate degree. In the past six years he had been admitted eight times, each time because of some diabetic complication; at times in insulin shock, but most frequently the ketosis could be traced to intercurrent infections, dietary indiscretions or alcoholic debauches. About four months before admission he was seen in the otologic outpatient department with the mildest type of acute otitis media. Although advised to return, he did not make his appearance until three months later, when he had a moderate amount of purulent discharge, some tenderness over the mastoid tip and a fairly innocuous appearing middle ear. Because of the known severity of his diabetes and the history of otitis four months before, we felt that he should be hospitalized. Roentgenograms of the mastoids revealed "some slight destruction of cells in the squamous region." Culture of pus was reported as showing Streptococcus viridans. A simple mastoidectomy was performed, and a perisinus abscess found. The patient's postoperative course was uncomplicated. He was discharged at the end of two weeks.

After three weeks at home the patient returned to the otologic service with the history that nine days before he had a temperature of 103 F. with severe anorexia. During that time he did not take any insulin, or eat anything. Examination at the time of admission revealed a profuse discharge from the middle ear and the postauricular wound. Two days later pain deep in the left eye, left-sided headache and tenderness over the left zygoma, with some edema, developed. Roentgenograms of the petrous pyramid and mastoid showed cloudiness over the left side as compared to the right. The left zygoma was somewhat cloudy as compared to the right, but no frank evidence of breaking down could be determined.

An exploration was deemed advisable. The old mastoid cavity was clean; the plate of the middle fossa was intact; the posterior root of the zygoma was found to have been cleaned out. With a small curet a pinhole cell in the zygoma was opened; then, on pressure over the swelling on the face, thick pus welled into the wound. More cortex was removed, and the site of perforation into the soft parts could be demonstrated. It was decided to explore the middle fossa further. The plate of the middle fossa was removed from behind forward. Far forward in the region of the middle root of the zygoma the dura did not have a normal luster. With a flat blunt elevator the dura was gently elevated; there was a gush of green pus. The plate was widely removed and normal dura exposed about the epidural abscess. The wound was thoroughly drained. The patient was quite sick following the operation, but gradually improved.

Comment.—The dichotomous spread of infection is definitely demonstrated in this case. Bearing in mind the known lack of resistance to infections characteristic of patients with uncontrolled diabetes, the severity of the process occasioned little surprise. Besides spreading externally and perforating into the soft parts and into the infratemporal

fossa, the process spread to the root of the zygoma and developed an epidural abscess. In retrospect, one realizes that in this case it was even more vital than ordinarily to exenterate all diseased cellular structure at the original operation, in order to forestall serious complications and secondary operations.

CASE 5.— J. L., aged 50, was operated on at a private sanatorium for acute mastoiditis. He did poorly; the wound did not heal, and a swelling developed preauricularly. This was incised, and pus was obtained. The patient was finally referred to the otologic service of Dr. Friesner, where he was reexplored. A large, suppurative, carious process was encountered in the zygoma. A large epidural abscess was found in this region. He did nicely after this procedure; all symptoms disappeared; the discharge lessened definitely, and the patient was discharged improved. Three weeks later he was readmitted to the otologic service with evidence of meningitis. A spinal tap revealed fluid under increased pressure, with a pleocytosis of 2,000. No organisms were found on smear or culture. The neurologic status at this time was negative. Again the patient was reexplored.

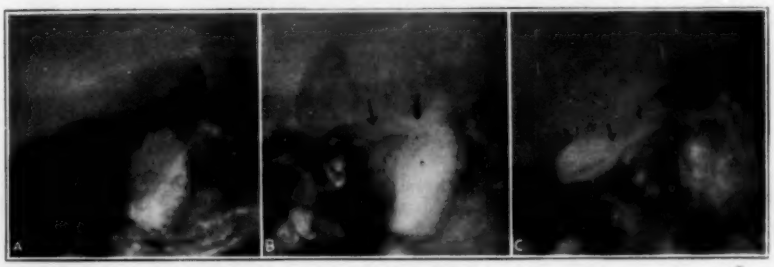

Fig. 3.—*A*, x-ray picture taken on admission to the hospital, showing extent of cell exenteration after initial operation. *B*, x-ray picture taken after cleaning out a large carious focus in the zygoma and an epidural abscess. *C*, three weeks later the patient was readmitted with meningitis. He was reexplored. This picture indicates the extent of dissection. Complete recovery took place after this.

The incision was carried upward and forward around the auricle. At a distance of about 1¾ inches (4.37 cm.), softened necrotic zygomatic bone about the size of a dime was removed. The dural plate was removed, and granulations were noted on the dura, but no pus was seen. The zygoma and the wound were kept open. In two days the meningeal signs disappeared entirely. Gradually, the discharge lessened, the middle ear became dry, and the preauricular fistula closed. The postauricular wound healed. During the period of convalescence, a focus developed in the spine.

Comment.—This case illustrates the seriousness of the intracranial complications that may result from an unhealed focus in the zygoma, i. e., meningitis with 2,000 cells in the spinal fluid. Nevertheless, with proper exenteration of the disease process the meningitis was checked. It also illustrates the futility of merely incising the abscess externally over the face without further treatment of the focus in the bone. It is imperative, whenever possible, to attack the disease process from the

original site of infection, namely, from the wound in the mastoid. The roentgenograms in this case are of particular interest in that they show the extent of the infectious process in the zygoma and the limits reached at each operation (fig. 3).

CASE 6.—A. G., aged 6, was admitted to the pediatric service twice, each time with a discharge from the ear and meningeal signs. On the first admission, cloudy, turbid spinal fluid was obtained, with a marked pleocytosis but no organisms. He was treated for meningococcus meningitis and was given serum. He made an apparently uneventful recovery.

Six months later he was again admitted with a history of ten days' duration, beginning with pain in the left ear. Myringotomy was performed, and pus was obtained. One day prior to admission, the patient had headache and vomited. On admission, there was evidence of meningeal irritation, and again a pleocytosis in

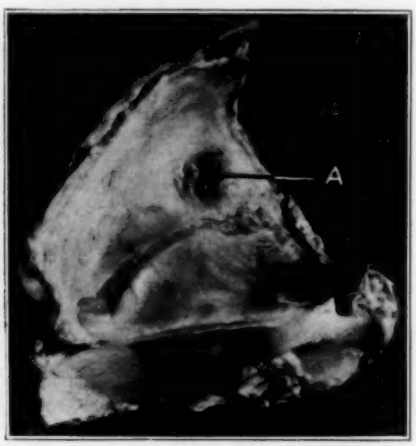

Fig. 4.—Gross photograph of the temporal bone, showing a large area of necrosis of the bone at the inner aspect of the root of the zygoma (*A*).

the spinal fluid with 67 per cent polymorphonuclears but no organisms was found. The fever subsided and the ear began to resolve, but despite this, there was a progression in the drowsiness, headache and papilledema. The consulting ophthalmologist noted a homonymous hemianopia. The focal signs became marked; aphasia developed, and the patient died before exploration could be performed.

The postmortem findings and the histologic sections revealed the following: "Situated in the left spheno-parietal lobe of the brain immediately under the cortex was a large abscess cavity filled with greenish pus and exudate; the abscess cavity was covered on its surface by a thickened capsule of fibrous tissue." Corresponding to the root of the zygoma was a small area about 1 cm. in diameter, in which denuded dura and periosteum were evident. There were adhesions between this lesion and the capsule of the abscess of the brain (fig. 4). Serial histologic sections revealed evidence of healing in the mastoid and direct extension of the disease process from the mastoid antrum to the root of the zygoma directly through the zygoma to the meninges and brain (figs. 5 and 6).

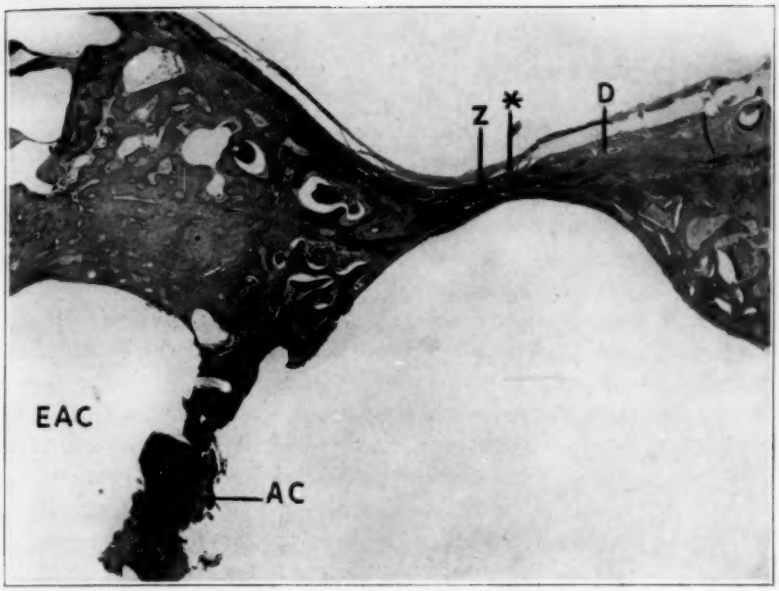

Fig. 5.—Vertical section through the root of the zygoma, showing purulent infection in the zygomatic cells with necrosis of the bone. The overlying dura is markedly thickened and inflamed. *AC* indicates the anterior canal wall; *EAC*, the external auditory canal; *Z*, the zygoma, and *D*, the dura.

Fig. 6.—Photomicrograph; higher magnification of figure 5, taken at *, showing marked necrosis of the zygoma, bony sequestration, thrombosed blood vessels and thickened dura. *B* indicates sequestration of bone; *PE*, purulent exudate; *V*, a thrombosed vessel; *D*, the dura.

Comment.—This case, like the last one, indicated the severity of the complication that may follow zygomatic involvement, i. e., an abscess of the temporal lobe which was the cause of death of the patient. An abscess of the brain secondary to the zygomatic disease is indeed rare, only one other case having been reported previously (Hutchinson and Greenwald, quoted by Lewandowsky [9]).

The infection spread insidiously from the middle ear and antrum to the zygomatic cells, subsiding behind but increasing in severity as it progressed, until finally it perforated at the inner aspect of the zygoma. Histologic sections showed slight changes in the tympanum and antrum. The character of the progressing severity of infection in its course was similar to that noted in our cases of osteitis of the petrous pyramid.[10]

This case is also of interest clinically in that no clue was offered as a diagnostic aid in determining the involvement of the zygoma. The middle ear had practically resolved; there was no evidence of a mastoiditis, nor was there any swelling or pain over the zygoma.

SUMMARY AND CONCLUSIONS

The anatomic and histologic structure of the zygoma, together with the signs and symptoms are discussed. Six cases of zygomatic involvement are presented. In four of these cases the symptoms became evident after mastoidectomy. In one they were present prior to the operation, while in one there was no gross evidence of any disease in that structure, the diagnosis being made on postmortem examination.

It is our belief that a more definite comprehension of the anatomy of the zygomatic process of the temporal bone will aid the operator in following the disease process into the posterior and anterior root to the limit, and thus frequently forestall many of the latent complications that might ensue, namely, malunion, persistent postauricular fistulas, epidural abscess, abscess of the brain and meningitis.

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9. Lewandowsky, M. H.: Der Hirnabscess, Handbuch der Neurologie, Berlin, Julius Springer, 1910-1914, vol. 3, p. 1909.

10. Friesner, I., and Druss, J. G.: Osteitis of the Petrous Pyramid of the Temporal Bone Associated with Paralysis of the External Rectus, Arch. Otolaryng. **12**:342 (Sept.) 1930.

CAVERNOUS SINUS THROMBOSIS FOLLOWING SUBMUCOUS RESECTION

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Submucous resection is generally regarded as a harmless, simple procedure. Nevertheless, the medical literature issues, not without infrequency, a slow, unending stream of reports of complications following submucous resection. The commonplace complications are familiar to the rhinologist and require no further exposition. More unusual sequelae, sources of infinite worry to the surgeon, are generalized septicemia, meningitis, hemorrhage, abscess of the brain and erysipelas. To this may now be added cavernous sinus thrombosis. Although it seems certain that this complication must have been reported, a careful search of the literature of the past twenty years fails to disclose the report of a case similar to mine. Consequently, I feel justified in stating that cavernous sinus thrombosis is a rare complication following submucous resection.

REPORT OF A CASE

History.—S. C., a man, aged 33, was first admitted to our outpatient department on April 9, 1932, complaining of frequent colds, annoying blockage of the left naris and headaches. This difficulty began suddenly two years before admission.

Examination.—Routine examination showed a septum markedly deviated to the left, with almost complete occlusion of the nasal passageway; the teeth were apparently in good condition; the tonsils had been thoroughly removed, with well preserved pillars, and the larynx was essentially normal. Both ear drums exhibited normal findings. Transillumination of the antrums and the frontal sinuses showed that the sinuses were normally translucent.

A submucous resection was advised at this time.

Operation and Course.—On July 9, 1932, a submucous resection was performed. In preparing the patient, the outside of the nose was cleansed with alcohol, and the vestibule was wiped with a 1:5,000 solution of mercuric chloride. The ends of four wooden applicators, immersed in a 10 per cent cocaine solution, were placed so as to block the anterior nasal nerves and those from the sphenopalatine ganglion. After fifteen minutes, a small amount of a 1 per cent solution of procaine hydrochloride (with 5 drops of epinephrine to the ounce) was injected under the mucosa of the septum. No particular technical difficulty was encountered during the operation, and the usual submucous technic was followed.

From the Ear, Nose and Throat department, service of Dr. Francis L. Lederer, Research and Educational Hospitals, University of Illinois, College of Medicine.

Two strips covered with petrolatum were inserted lightly in the nose, and the patient was sent back to his room in good condition. He remained in the hospital for two days following the operation, according to our routine. The strips were removed after twenty-four hours. An ice bag was kept constantly to the patient's nose. The postoperative recovery, at this stage, was uneventful. The nose, at the time of the patient's dismissal, was clean and dry, and the mucous membrane flaps remained in approximation.

Two days after the patient's discharge from the hospital, he returned to the outpatient department, complaining bitterly of a severe headache which had awakened him that morning and had persisted throughout the early part of the day. When the boggy mucous membrane surface was shrunk with epinephrine, no sign of infection was revealed. The flaps were still approximated. On the next morning, the patient returned with the same complaint, and as a precautionary measure he was hospitalized. On admission, his temperature was 100.2 F.

Patient twenty-four hours before death.

For the next three days the patient's condition was mildly septic, characterized by excruciating intermittent headaches partially relieved at first by acetylsalicylic acid and amidopyrine, U. S. P., and later by opiates. The nose was shrunk periodically by the use of epinephrine tampons. Physicians from the neurologic, medical and ophthalmologic departments were called into consultation. Their findings were essentially negative and confirmed our own. Urinalysis and chemical examination of the blood gave no clue, although a differential blood smear showed 90 per cent of polymorphonuclears. A roentgenogram showed a slightly increased density over the right antrum.

On July 17, the temperature mounted to 101 F., and the patient complained of pain in the left eye. There was some associated edema of the lids on both sides. Two hours later the left eye was proptosed, and a diagnosis of cavernous sinus thrombosis was made. At 1 p. m. on that same day, the right eye became proptosed, and the picture of bilateral cavernous sinus thrombosis was now complete. Mentally, the patient became listless and sluggish in his replies. Both eyes were markedly proptosed with chemosis, and the edema was well established

(illustration). There was a rotatory nystagmus with pronounced limitation in the excursion of the external recti muscles. No tenderness was elicited on palpation over the frontal sinuses or antrums. The fundi gave no findings other than a suggestive pallor at the temporal borders of the disks. The nose continued clean in appearance and devoid of secretion. There was moderate tenderness over both jugular veins. Incubation of the blood culture for two days revealed hemolytic staphylococci. Late that night the patient became semicomatose. Septic pneumonia and terminal meningitis preceded death the following morning.

Autopsy.—The meninges were markedly adherent, the pia arachnoid deeply injected and the convolutions flattened. At the pons and extending to the optic chiasm was a thick fibropurulent exudate. The transverse sinus contained a small amount of recently clotted blood. Serial section of the brain revealed no pathologic condition. The right lateral sinus was filled to and beyond the jugular foramen by a soft, friable, dark red clot. The cavernous sinuses on both sides were occupied by thick, purulent, sanguineous material. The ethmoid and sphenoid sinuses on both sides were filled with thick creamy pus which, in some of the cells, was mixed with blood.

Other incidental findings were bronchopneumonia, with multiple embolic abscesses in the lungs; a primary tubercle in the right lower lobe; a hemorrhagic ulcer in the pyloric region of the stomach, as well as a healed pyloric ulcer; cloudy swelling of the liver and kidneys, and a septic infarct of the spleen.

Comment

A review of the anatomic details of the cavernous sinuses is pertinent. The cavernous sinuses are paired venous sinuses lying on both sides of the sella turcica on the body of the sphenoid bone, directly under the middle portion of the cerebrum and almost directly posterior to the inner canthus of each eye. Posteriorly, they terminate in the superior and inferior petrosal sinuses, and measure about 2 cm. in length anteroposteriorly. Anteriorly, they receive the valveless superior and inferior ophthalmic veins, which in turn anastomose freely with the angular, frontal, supra-orbital and facial veins, also valveless. Branches are also received directly or indirectly from the cerebrum, pterygoid plexus, ethmoidal veins, sphenopalatine veins, pharyngeal plexus, posterior auricular occipital veins, vertebral sinuses and basilar plexuses.

In front of and behind the sella turcica are the communicating sinuses connecting the right and left cavernous sinuses. Thus a circular sinus is formed about the upper aspect of the sphenoid bone. The cavernous sinuses are trabeculated or honeycombed by numerous thin, membranous splits from the dural walls. Lying within each sinus is the internal carotid artery and the sixth cranial nerve, and along the lateral aspect, embedded in the dural walls, lie the third and fourth cranial nerves and the ophthalmic and superior maxillary divisions of the fifth nerve. The sphenoid air sinus lies directly under the cavernous sinus on both sides and is separated from it by the thin, bony wall of

the sphenoid and the dura. Occasionally, a posterior ethmoid cell extends backward into the body of the sphenoid bone so as to encroach on the area more commonly occupied by the sphenoid air sinus. Thus the superolateral wall of an ethmoid air cell may be contiguous to the cavernous sinus.

Turner and Reynolds,[1] in a summary covering twenty-two instances of septic cavernous sinus thrombosis, state that the pathways of infection were as follows: In thirteen cases, infection reached the sinus entirely by way of its afferent or efferent venous channels from the face, pharynx, air cavities and the cleft of the middle ear; in eight, the preliminary stage of the infective process was direct extension through the bone, followed by further spread to the sinus along osseous veins; in the latter group, the primary focus was in the frontal, ethmoidal or sphenoidal air sinuses. In the remaining instance the infection passed to the blood sinus from the sphenoidal air sinus entirely by direct extension of the inflammation through the bone and dura mater.

Infection of the cavernous blood sinus arises most frequently as a complication of a peripheral septic focus situated in some part of the extracranial area with which its afferent and efferent vessels communicate. When infection reaches the sinus, a small thrombus forms in the interior of one of the blood spaces. The clot eventually fills all the spaces. From this center the thrombotic process radiates progressively along both the tributaries and the efferent channels of the sinus. In the afferent channels the thrombus grows in the direction contrary to the normal current of the blood stream. This is the so-called retrograde formation of a thrombus. Along the efferent vessels, however, the clot extends in the same direction as the normal flow of blood.

Its extension along these channels is the etiologic factor producing the pathologic processes and clinical manifestations characteristic of the picture of the disease. Consequently, with the retrograde spread of the thrombus into the ophthalmic veins there arise edema of the eyelids, chemosis and exophthalmos and dilatation of the retinal veins; into the superficial cerebral veins, the frequently associated secondary purulent leptomeningitis and sometimes, too, an area of localized abscess or cerebral softening spread. The extension of the clot along the efferent channels may, in the progress of the disease along the basal emissary veins, give rise to a pharyngeal abscess; along the superior petrosal sinus and transverse sinus, to retro-auricular edema and occipital abscess, and with the involvement of the jugular vein, to pyemic abscesses in various organs of the body.

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1. Turner, A. Logan, and Reynolds, F. E.: Intracranial Pyogenic Diseases, Edinburgh, Oliver & Boyd, 1931.

Sooner or later, the septic process implicates the intercavernous venous plexus, with extension of the clot into the cavernous sinus of the opposite side. Thus the condition, at first unilateral, becomes bilateral with the reproduction, on the opposite side, of the same pathologic changes with or without clinical signs more or less identical with those on the side first affected.

CONCLUSIONS

A period of two months had elapsed between the initial appointment with the patient and the day of operation. As frequently happens in overcrowded, overworked dispensaries, the patient was not seen during this interim. A history of swimming and a cold was obtained later from the family. In commenting, it may be well to emphasize the importance of adequate and frequent preoperative examinations of the patient.

In this case, while no actual route of invasion could be demonstrated at autopsy, most probably infection occurred by extension through the ethmoidal veins. It is possible, however, that there was a direct extension by continuity of tissue from the sphenoidal air sinus directly into the cavernous sinus, although there was no demonstrable dehiscence of the upper sphenoidal wall.

A careful review of the literature on the subject establishes the fact that surgical procedure directed at drainage of the cavernous sinus is hardly justifiable in view of the difficulty of approach and the multilocular nature of the sinus. In favorable cases seen early, and in the abscence of meningitis, surgical intervention may sometimes prove beneficial.

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THE PROBLEM OF MENINGEAL INFECTION FROM PETROSITIS

REPORT OF CASES

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Despite the effort of distinguished surgeons in many quarters, surgical treatment of otitic leptomeningitis yields but meager results. Several commendable clinical and pathologic studies [1] have appeared in recent otologic literature to demonstrate clinical variations, prodromal symptoms, mechanism of meningeal contamination from petrous disease, manifestations of otitic leptomeningitis and suggested surgical relief. With the basic knowledge supplied by these studies, the alert otologist will recognize early symptoms and occasionally save a patient from this costly complication of otitis media.

As there is no rational generally accepted plan of surgical treatment which can be depended on to save even a fair percentage of these patients, it is important that those dealing with this problem pool the results of their studies until prodromal symptoms may be more quickly and accurately recognized and specific clinical and surgical measures applied specifically to early contamination of the meninges before the basal cisterns are involved. Pneumatization in the petrosa as well as in the mastoid process should be demonstrable by a standard recognized roentgen technic, and such facilities should be available in contagious sections, where these problems most often arise, especially as a complication of scarlet fever. A comparison of early and late roentgen studies is often of great value.

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1. (*a*) Kopetsky, S. J., and Almour, R.: Suppuration of the Petrous Pyramid; Pathology, Symptomatology, and Surgical Treatment: I. Introduction: Anatomic and Pathologic Data, Ann. Otol., Rhin. & Laryng. **39**:996 (Dec.) 1930; II. Symptomatology, ibid. **40**:157 (March) 1931; III. Surgical Therapy, ibid. **40**:396 (June) 1931; IV. Case Reports and Pathologic Data, ibid. **40**:922 (Sept.) 1931. (*b*) Eagleton, Wells P.: Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926; Unlocking the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, Arch. Otolaryng. **13**:386 (March) 1931; Suppurative Meningitis of Otitic and Nasal Origin, ibid. **15**:885 (June) 1932. (*c*) Profant, H. J.: Gradenigo's Syndrome, with a Consideration of Petrositis, Arch. Otolaryng. **13**:347 (March) 1931.

The difference between osteomyelitis and empyema must be recognized, because different treatment is indicated. In osteomyelitis sequestrums form, but often in empyema of the coalesced tip there are no sequestrums. There may be a coalescence of a pneumatized tip without evident mastoiditis. No operation on the mastoid tip is adequate to eliminate infection within the tip if the pyramid is involved and the ear continues to discharge.

Attention has been called to the fact [2] that when well developed air cells are present in the petrosa their development increases the space relationship between surgically important structures. If cells develop from the anterior region of the eustachian tube and zygomatic root, there is increased space between the knee of the carotid artery, the facial nerve, the semicircular canals, the cochlea and the superior wall of the petrous pyramid. If cells develop along the posterior wall, there is more distance to the labyrinth capsule. This should offer better hope of providing necessary drainage of pus under pressure when it is traced into the pyramid and before it becomes an endocranial infection. Practically, this is not always true, as demonstrated in my second case. The middle ear at operation is usually full of detritus. Theoretically, surgical intervention should give relief. Practically, the mortality has been little changed by it. Because of this fact, various workers have been investigating fundamental changes in the pathologic anatomy. Most recent investigators believe that the spinal fluid is a dialysate, a filtrate from the capillaries of the choroid plexus and, in a small amount, from the capillaries of the perineural and perivascular spaces of the central nervous system. This fluid is normally in osmotic equilibrium with the blood so that intravenous injection of a hypertonic solution reduces intracranial pressure and diminishes the rate of formation of cerebrospinal fluid. The ventricular and subarachnoid systems perform separate and distinct functions. The greatest volume of the cerebrospinal fluid is formed in the lateral, the third and fourth ventricles. In the subarachnoid system of communicating spaces the ventricular fluid is distributed to the cortical surfaces, and ultimately leaves through the large venous sinuses. The cerebrospinal circulation is from the lateral ventricles through the foramina of Munro and the third ventricle, through the aqueduct of Sylvius to the fourth ventricle, and then through the foramina of Magendie and Luschka to the cisterna magna. The fluid is so distributed to guard various parts of the brain from shock and injury. It has been concluded [3] from a composite of many

2. Lilly, H. I., and Williams, H. L.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **15**:692 (May) 1932.

3. Kerrison, Phillip D.: Treatment of Otitic Leptomeningitis, Ann. Otol., Rhin. & Laryng. **41**:651 (Sept.) 1932.

pathologic reports that of the various subarachnoid cisterns those most frequently involved in otitic meningitis are: the cisterna interpeduncularis, the cisterna chiasmatis and the cisterna fissurae sylvii. From their frequent and extensive involvement, infection of these spaces exerts a striking influence on the spread of basal infection and the high mortality.

Davis,[4] in a report to the British Medical Society, presented pathologic reports in twelve cases. In all there was a large collection of pus between the tentorium and cerebellum and pus in the cisterna interpeduncularis. The optic chiasm in each contained pus, i. e., the cisterna chiasmatis contained pus. In more advanced cases the infection spread along the sylvian fissure to the cerebral cortex. The cisterna magna was free from pus in some cases, and not markedly infected in any. As these cisterns are intercommunicating, it would seem possible to treat them surgically as one infected cavity. Nature's first response to cerebrospinal infection is an increase in the amount of cerebrospinal fluid to dilute the toxins. This accumulates in the basal cisterns, and circulation is stopped by its own back pressure. In severe inflammatory conditions, the various compartments become walled off. When this has happened, it is impossible to establish any really adequate drainage from a single point of outflow. It is essential to establish drainage as early as possible and drain from the deep cisterns as well as from the surface. Wide opening of the brain or spinal column allows collapse of the meninges, with resultant partial obstruction, which will prevent drainage for which the opening is made. This may be prevented by forcing new formation of fluid, by lowering the osmotic pressure of the blood and allowing free escape of cerebrospinal fluid, which washes the ventricular and arachnoid systems and works the infection from the depths to the surface if free drainage can be maintained. Such drainage combined with elimination of the active focus of infection in the petrous bone offers some hope of recovery. If regions become blocked, Kerrison packs off the lateral sinus and drains the larger cisterns through its inner wall. Slow intravenous injection of hypotonic solution may increase cerebrospinal fluid more than ten times. There are certain obvious limitations. The gelatinous thick exudate in some forms of pneumococcus infection is always a problem, as are also encapsulated foci and pockets. This method has not as yet been subjected to adequate clinical trial. One may occasionally use the homolateral sphenoid for drainage of an apical abscess. The direct surgical approach to eliminate the primary infection has been admirably described by Eagleton, Kopetsky and Almour.

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4. Davis, E. D. D.: Paper read before the British Royal Medical Society in 1921.

I wish to present four cases which will review the problems encountered.

REPORT OF CASES

CASE 1.—*Perilabyrinthine peritubal cell petrositis.*

History.—In M. H., aged 29, sudden, severe otitis media developed on the right side during scarlet fever. There was associated pseudomembranous nasopharyngitis. A prompt myringotomy was followed by a profuse serosanguineous discharge. On the twelfth day, in the midst of an apparently normal convalescence, there recurred an afternoon fever. There were no symptoms of mastoiditis. The right external canal was still filled with mucopurulent drainage. On the fourteenth day, the afternoon temperature rose to 102.5 F. When asked to sit up, the patient stated that she felt dizzy and uncertain. Shortly after this, a fine, rapid spontaneous nystagmus was noted. Preparation was made for operation immediately. At the time of operation there was a slight rigidity of the neck and a spinal fluid cell count of 2,500, with cloudy fluid. A simple mastoidectomy was performed, the incision being made high, far forward, and deep enough to obliterate well all mastoid cells and provide an apparently sufficient external perilabyrinthine drainage. Ten cubic centimeters of Pregl's solution (resulting from the action of iodine on sodium carbonate solution) was injected into the right carotid to flood the infected meninges with nascent iodine. Spinal drainage was done at frequent intervals, and at the least sign of increasing restlessness. This required the constant and devoted attention of two commendable interns. Intravenous injections of 50 per cent dextrose were given every four hours, and also caffeine sodium benzoate in doses of 7½ grains (0.49 Gm.). Nasal gavage was necessary to sustain the patient. From 15 to 20 cc. of Pregl's solution was injected daily into alternating carotids by a fine sharp needle puncture. After the iodine solution was administered, the spinal fluid was sterile, and the few organisms seen at times on smear would not grow on culture. The highest spinal fluid count was 6,050, which fell after fourteen days to 60, after which it was not taken because of the excellent condition of the patient. She read the newspapers and was much interested in the welfare of her four small children. On the twentieth day, headache and spinal root pains recurred; a typical basilar meningitis developed which failed to yield to all measures, including cistern puncture.

Pathology.—The spinal root pains were explained by the finding that the lower thoracic and lumbar portion of the spinal cord showed a reddish-gray exudate on the external surface of the dura; when the dura was opened there was a thick, greenish-gray exudate, most marked in the lumbar region, and this followed the exit of the roots through the dura. The petrous bone was very cellular, particularly about the labyrinth, the cochlea and the peritubal region. On the anterior surface half the distance to the apex was a 5 by 8 mm. defect, the apparent source of reinfection. There was an extensive infection of the basal cisterns. The cortex showed only a moderate amount of yellowish-gray exudate.

Comment.—When many spinal drainages are necessary in the presence of infected fluid, the surface must be kept dry and clean. The smallest caliber needle that will give efficient drainage is of great advantage. Had the petrositis resolved by adequate external drainage, the defect would not have developed and a complete recovery might have resulted. With the added knowledge of petrositis that has been made available within the last two years, a more radical approach to the

petrosal cells would be made in a similar case. It is significant that the spinal fluid became sterile promptly after intracarotid injection of Pregl's loosely combined iodine solution. With the striking clinical improvement in this case, it was difficult to find reason to reoperate. With the sudden recurrence of symptoms, it was too late.

CASE 2.—*Peritubal hypotympanic route petrositis; apical abscess.*

History.—G. I. B., aged 6, entered the hospital with scarlet fever, suppurating right cervical glands and developed bilateral otitis media. A bilateral myringotomy was done. After four weeks, with increasing otorrhea and clouded and tender mastoids, a bilateral, simple mastoidectomy was done. Two weeks later the patient was dismissed as cured and returned to his home. He remained free from fever for two weeks. There then developed low recurrent afternoon fever, recurrent otorrhea on the left side, diplopia with paralysis of the left external rectus muscle, left-sided headache, pain behind the eye and pain over the left antrum. These symptoms were maintained for eight days. On the ninth day, the temperature rose sharply, and the patient was readmitted for further exploration of the left temporal bone. The perilabyrinthine cells were thoroughly cleaned out. The middle ear cavity was filled with the customary débris. The technic of Kopetsky and Almour was as yet unpublished, and no attempt was made to attack directly the peritubal cells for drainage. After forty-eight hours, typical basilar meningitis developed, and after seventy-two hours, the patient died.

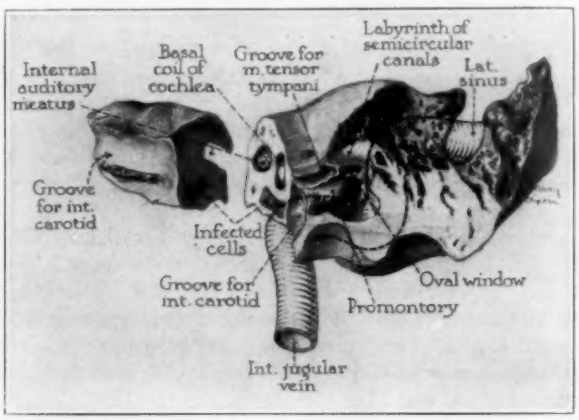
Pathology.—The right temporal bone was normal. There was a typical basilar meningitis. There was soft pinkish granulation tissue causing adhesions between the dura and the body of the sphenoid bone on the left side. This extended centrally to involve the posterior portion of the bone in the region of, and eroding, the posterior clinoid process. There was a deep jagged cleft at the medial extremity of the petrous portion of the temporal bone extending laterally on both the posterior and the anterior surfaces and filled with thick yellow pus. This cleft was lined with pink, rather tough, granulation tissue.

Comment.—Almour [5] would have converted the cavity in the mastoid into a radical and burred off the anterior wall and zygomatic root to expose the mouth of the eustachian tube fully. The tensor tympani muscle and its bony encasement would next be removed, and a 1.5 mm. burr placed at the level of the musculotubal canal 1.062 mm. posterior to the mouth of the eustachian tube and directed at a 22 degree angle to the external canal. This is followed by curettement of the cavity and the placing of a rubber drain. In the drawing of the temporal bone from this patient it is to be noted that the space relationships are not increased, the surrounding bone is ivory hard and the offending abscess is far away, with only a tiny tract of infected cells. The chances of providing adequate drainage in this instance by this method are remote. The location of this apical abscess might have made it amenable to homolateral sphenoid drainage. The illustration demonstrates the unusually solid character of the petrosa except for the tiny cell tract which led to the apex to form an apical abscess and produce erosion of the sella turcica.

5. Footnote 1a and b.

CASE 3.—*Diploic structure generalized osteomyelitic petrositis.*

History.—H. J., aged 15, was ill for one week with influenza. During this time otitis media developed on the left side, which ruptured spontaneously, drained three days and healed. The patient was a high honor high-school student, and after ten days from the onset of her illness returned to school for the entire examination week, stopping after seven days because of an increasing left-sided headache. I first saw her because of a severe, persistent, high, frontal pain on the left side. An examination of the ear at this time revealed a slight thickening of the drum, and a slight pinkish cast, with little bulging and no pain. Because of the history of aural infection earlier, a myringotomy was done for safety and only a small amount of clear serum exuded. All sinuses were clear. On the following morning there was increased left-sided headache, inability to retain food and a rising temperature. It was so difficult for the parents to understand the seriousness of the situation that one day was lost before drainage could be established by operative means. At this time the spinal fluid cell count was 1,600, and hemolytic streptococci were grown on culture. There was also paresis of the left external rectus muscle. A



Temporal bone

modified radical drainage was done, with special effort made to provide anterior and deep petrosal drainage. This was followed by three comfortable days. On the fourth day there was further evidence of basilar meningitis, and on the seventh postoperative day the patient died in a convulsion. Sustaining treatment was similar to that previously outlined.

Essential Pathology.—The tip of the right (undiseased) petrous bone contained bright red marrow in a healthy condition. The left (diseased) petrous bone broke over the inner table easily and revealed a brownish-gray, turbid exudate in the cancellous portion, extending out into the tip, as evidenced by the brownish discoloration and gelatinous appearance of the red marrow. An attempt to remove the temporal bone for further study was only partially successful because of the brittle diploic character.

The dura was moderately tense. The cerebral gyri were somewhat flattened equally on the two sides. The inferior surface of the frontal lobes showed a delicate film of fibrinous exudate and fairly numerous petechial hemorrhages. The temporal lobes showed similar exudate, hemorrhages and marked injection of the

smaller blood vessels. The surface of the pons, anterior and inferior surfaces of the cerebellum and the medulla showed a more abundant gray fibrinous exudate and innumerable petechial hemorrhages.

Comment.—This type of pathologic process offers the most difficult problem. Essentially, the base of the skull is unilaterally involved in an osteomyelitis, and simple drainage of a tract of cells does not suffice. If wide open treatment could be used, as in osteomyelitis of the long bones, fair hope of recovery could be entertained. Intracarotid therapy, flushing of the deeper cisterns by hypotonic solutions intravenously and other methods fail because of the impossibility of eradicating the primary infecting focus.

It is recognized that in many cases of the Gradenigo syndrome type recovery is either spontaneous or follows simple drainage of the mastoid. The three cases reported represent a definite extensive pathologic process and not secondary edema effects.

CASE 4.—*Petrositis; cortical meningitis, self-limiting arachnoiditis; recovery.*

History.—In W. H. Z., aged 12, otitis media developed on the right side during scarlet fever. At the end of the fourth week of otorrhea, typical symptoms of mastoiditis developed. At operation, a very cellular structure was encountered, so the cells were followed far forward into the zygoma and deep into the petrosa. Forty-eight hours later, symptoms of meningitis developed. There was a positive Kernig sign, a stiff neck, and the spinal fluid cell count was 950. No organisms could be cultured at this time. There was marked mental instability and crying, and the patient could not be reasoned with. He was sustained by nasal gavage and the usual supportive treatment for meningitis. On the fourth postoperative day there was weakness of the left facial nerve and weakness and decerebrate position of the left hand. These were the first motor symptoms. On the fifth day there were convulsive attacks affecting the left eyelids and the jaw muscles, so that it was necessary to use a mouth gag to prevent laceration of the tongue. On the sixth day the left arm was also involved during the convulsive attacks. On the two succeeding days the entire right motor area was apparently involved in the irritative process, and the entire left side of the body was involved in the convulsions. On the ninth day there was evidence of spread to the opposite side. On the afternoon of the tenth day I was informed that the temperature of the patient was 107 F., that a convulsive attack involving the whole body, slightly less in the upper right portion, showed no tendency to abate, that the pulse was very rapid and the color bad. Sodium amytal administered intravenously was immediately ordered, and after a quantity of solution equivalent to 10 grains (0.65 Gm.) had been injected over one and one-half minutes the patient was quiet and asleep. In one hour the temperature was 103 F. All convulsions thereafter were promptly terminated with intravenous administration of the minimum dose of sodium amytal required. As the meningeal infection receded and the cortical irritation was relieved, the convulsive movements in the muscle groups involved receded in exactly the reverse order of onset. After a prolonged convalescence of several weeks in the summer sun, the patient returned to his school for the autumn session.

Probable Pathology.—I have classified this case as one of petrositis because of the operative findings. In all probability, the infection, so far as organisms were

concerned, was limited by a protective arachnoid barrier. Such lesions have been described. Anatomically, one cannot demonstrate cortical arachnoid compartments, but clinically such self-limiting compartments may result from infection.

Comment.—A wide, satisfactory, early external drainage, together with the timely intravenous use of an effective hypnotic, were undoubtedly the two life-saving measures. The reason for the extraordinary cortical course with no important involvement of the basal cisterns is not readily explained on the basis of the petrositis, which usually produces a basilar meningitis.

SUMMARY

Case 1 represents a particularly cellular type of petrous bone with an inner table defect, an apparent clinical recovery, reinfection and surgical failure.

Case 2 represents a very solid ivory-hard type of temporal bone in a 6 year old child with a tiny tract of anterior, peritubal cells leading to the tip, abscess at the tip and osteomyelitis of the sphenoid body, with destruction of the posterior clinoid process. It illustrates the probable futility of a technic such as that described by Almour when used in this type of temporal bone after symptoms of the petrous tip have developed.

Case 3 represents still another type of pathologic process in the temporal bone with the homolateral half of the base of the skull involved in an extensive osteomyelitis of the diploic structure. Surgical treatment is doomed to failure without the development of some wide open technic such as is used in the surgical procedure for the osteomyelitis of the long bones.

Case 4 represents a surgically demonstrated petrositis with a cellular structure similar to that in case 1. The meningeal infection was of cortical character, and did not involve the base.

CONCLUSIONS

Anatomic variations in petrous bone structure with similar clinical pictures make the problem of the treatment of petrositis a particularly difficult one. One hopes that methods of surgical attack may yet undergo many refinements. One may even anticipate that confrères in immunology may aid tremendously in these problems of inaccessible infection by being able to attack identified organisms by specific serologically immune or antitoxic administrations. For the present, it is highly important to attack the lesion by early surgical intervention with skilful thoroughness before there is endocranial infection, and particularly before the basal cisterns are flooded with organisms. After meningeal contamination develops, further clinical experience must prove whether it is wiser to use hypertonic solution intravenously to withhold

the formation of spinal fluid, or to use hypotonic solutions to increase greatly formation of cerebrospinal fluid and thus flush out the deeper cisterns where adequate drainage can be maintained. All indirect methods, such as intracarotid therapy, will continue to be ineffective when the primary infecting focus is not rendered inactive.

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CARTILAGE AND IVORY

INDICATIONS AND CONTRAINDICATIONS FOR THEIR USE AS NASAL SUPPORT

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In previous papers dealing with the use of ivory implants in rhinoplasty I stressed the importance of the morphologic and chemical structures of this substance with reference to its tolerance by human tissues.[1] To my knowledge these were the first communications in the medical literature pertaining to the use of ivory in reconstructive surgery.

During the eight years that have elapsed since the publication of these communications, I have continued to use ivory implants in selected cases, giving preference to cartilage grafts in most instances. Reports on this subject which have appeared in the medical literature in the last few years are far from uniform. Satisfactory end-results are claimed by a number of reliable observers,[2] while others point to the difficulties met with in shaping the implant or to infection following its introduction. The mere fact that ivory is not an autoplastic material seems to a great extent to prejudice its use, but its biologic merits, as well as reliable experimental and clinical observations, point decidedly to its usefulness in filling out defects, especially those of the facial skeleton.

The question may arise as to why it should be necessary to use a dead organic substance as support when it is generally conceded that rib cartilage is an excellent plastic material. Experience has shown, however, that in numerous instances, cartilage does not possess the required plastic qualifications, and in many cases it cannot be obtained. Clinical observation has proved that ivory implant is a good substitute for cartilage wherever there is a contraindication for rib cartilage resection or failure following its insertion.

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1. Maliniak, J. W.: (*a*) Comparative Study of Ivory and Organic Transplants in Rhinoplasty: Endo-Nasal Operative Technic of Some Nasal Deformities with Report of Cases, Laryngoscope **34**:882 (Nov.) 1924. (*b*) The Use of Ivory in Rhinoplasty, Arch. Otolaryng. **1**:599 (June) 1925.

2. Joseph, Jacques: Nasenplastik und sonstige Gesichtsplastik: Nebst einem Anhang über Mammaplastik und einege weitere Operationen aus dem Gebeite der äusseren Körperplastik, Leipzig, Curt Kabitzsch, 1931.

ADVANTAGES AND DISADVANTAGES IN THE USE OF CARTILAGE

The tensile strength of rib cartilage and its lack of absorption make it popular where a resistant support is required. It is definitely established that rib cartilage does not absorb in the course of time, and the only change in structure which it undergoes is a slight calcification. Exceptions are not rare, and I have observed rapid absorption of the graft in instances in which the rib cartilages appeared markedly calcified. The natural tendency of rib cartilage to bend often makes its use undesirable in instances in which a long straight graft is required. The support required in rhinoplasty for rebuilding of the nasal bridge must be straight when inserted and must retain its shape following repair (fig. 1).

Fig. 1 (case 3).—*A*, bulky nasal bridge five years after insertion of a tibia bone graft (done elsewhere) for correction of an old nasal fracture. The graft was inserted between the split nasal bones, which explains its lack of absorption. *B*, the bone graft was removed by me through a columellar incision, the nasal bones split in the middle line and the laterally deviated wide bony bridge narrowed and replaced in the middle line by infracturing the frontal maxillary processes. The interest in this case lies in the fact that the insertion of a bony or any other type of graft was unnecessary in the repair of this type of nasal fracture. The deformity shown in *A* was caused by the insertion of the bone graft. Orthopedic methods are not always suitable in rhinoplasty.

It is difficult to foresee in what instances postoperative bending of the graft will occur. Attachment of the perichondrium predisposes bending of the cartilage, but it has been my experience that even after a thorough removal of the perichondrium, curvature of the graft is apt to occur, in spite of its perfectly straight shape when inserted and the the most rigorous postoperative immobilization. This tendency is less

frequently observed when the graft is of equal thickness throughout, and this can be obtained by its resection with a well balanced angular knife. The graft required is usually narrow and thin at both ends and thick in the middle part, where its tensile strength is also the highest. Bending is rarely observed in the graft inserted into the columella for support of the nasal tip as it is generally of uniform thickness. Although there is no particular difficulty in at least partially straightening the graft at its ends by secondary correction, the postoperative end-result in these instances is apt to be far from satisfactory because of the lateral curvature of the graft.

For reconstruction of a nasal depression involving the bridge, as well as the tip of the nose, an angular graft united at the knee by a strip of perichondrium is often recommended. In most instances, however, it is difficult, if not impossible, to procure a straight support of the required length (about 9 cm. in adults) from the normally curved rib cartilages.

The graft prepared under these circumstances is almost certain to bend. Separate supports for the dorsum and columella, in my opinion, are more suitable in these cases.

Thus, even though no absorption of cartilage substance occurs in time, the fact that the shape of the graft frequently undergoes some modification is often a decided drawback to its use in rhinoplasty. These facts were stressed by me a good many years ago.[1a] The disfigurement that follows the bending and twisting of the cartilage is a matter of great distress to the patient as well as to the surgeon. In these instances an ivory implant is the best substitute for cartilage (fig. 2).

In addition to the curling of the cartilage, there are often other cases in which an ivory implant may be indicated, namely: 1. Nervous and hypersensitive persons often are unwilling to provide a graft from their rib cartilage because of fear of additional scarring of the chest and the prolonged hospitalization required (fig. 3). 2. In elderly persons a rib cartilage resection may be inadvisable.

Besides the ribs there are other sources of cartilaginous supply which are often used when a smaller amount of materal is required. Septum, alar and lateral nasal cartilages, as well as the auricle, can provide a substantial amount of material. It is not possible, however, to procure a graft from these sources such as is required for correction of the entire length of the nasal dorsum. Limited depressions of the facial skeleton can, however, be comfortably repaired by means of this accessory cartilaginous substance. The ease with which this source can be approached should not be the deciding factor in selecting the procedure of choice. The abundance of yellow elastic tissue in septal

Fig. 2 (case 1).—Curling of a cartilaginous graft inserted in a saddle back deformity secondary to infection in early childhood. *A*, the nasal deformity, corrected elsewhere, by insertion of rib cartilage which curled shortly afterward. *B*, same patient after insertion of an ivory implant through the old glabellar incision without disturbing the previously inserted graft. The patient was unwilling to undergo another rib cartilage resection because of failure of the first attempt at correction. *C*, x-ray film of nose taken six years after insertion of ivory. Nasal examination at that time showed good immobilization of the implant and unchanged shape.

Fig. 3 (case 2).—*A*, depressed nasal deformity in a man of 43, caused by trauma in childhood. The patient, a busy executive, was unwilling to provide his own cartilage and could allow only a few days for repair of the deformity. *B*, an ivory implant inserted through a columellar incision corrected the nasal depression involving the lower two thirds of the dorsum. *C*, x-ray film, showing position of ivory implant five months after its insertion; the upper end of the implant continues (not superimposed) on the nasal bones. The frontal processes of maxillary bones were fractured in a separate stage prior to insertion of the implant with the view of reducing the bony width. The line of fracture is clearly visible on the film.

cartilage makes for its certain absorption. Following the absorption of the septal cartilage, there often remains a sufficient amount of fibrous tissue to effect some elevation. In using this material, overcorrection is therefore indicated. Auricular cartilage, because of its concave shape, is best suited for the repair of limited depressions of an appropriate form. Stripped, however, of its perichondrium and divided into small fragments, auricular cartilage may also be used for the filling out of small nasal defects. The amount of material procured by alar or triangular cartilages is usually small, but it may be of advantage in the repair of minor depressions.

IVORY

A chemical analysis of true ivory shows that its composition is similar to that of human bone, both differing only in the amount of magnesium phosphate and calcium carbonate they contain (table 1). Ivory contains about 43 per cent organic matter, while dentin contains only 25 per cent. Ivory substance has the tensile strength essential to the permanency of shape and is of a morphologic structure similar to that of bone without showing any tendency toward absorption. Its chemical and morphologic similarity to human bone explains why it is a nonirritant to the tissues. Clinically, it has been proved in my experience for the last twelve years that ivory remains in the tissues if introduced aseptically and in proper shape. This also has been proved in cases in which ivory has been found unchanged in human structures for thirty years. The experience of Joseph with ivory implants does not leave any doubt as to its value as a supporting material.[2]

Pseudo-Ivory.—Strict differentiation of true ivory from the so-called pseudo-ivories is essential, as their behavior in the tissues depends on their composition. To the pseudo-ivories belong all types of substances which have the exterior aspect of true ivory without being of the same chemical composition. Vegetable ivory consists mostly of gum, legumin and lignin. Celluloid contains nitrocellulose (73.7 per cent) and camphor (22.79 per cent). With these facts in mind, it should not be difficult to understand the different reactions observed in human tissues toward these substances. With a structure so similar to bone, it is not surprising that ivory is readily tolerated, while the pseudo-ivories, being entirely foreign to the human structures, are ejected sooner or later (table 2).

A thorough differentiation of true ivory from pseudo-ivories is, therefore, essential. The cheapness of vegetable ivory is responsible for its frequent representation as true ivory, this being made possible by the external similarity. In treating each with concentrated sulphuric acid for fifteen minutes, vegetable ivory becomes rose-colored, while

true ivory remains white. This test should be made whenever there is the slightest doubt as to the authenticity of the ivory substance.

Preparation of Implant.—A plaster of paris or Negocoll cast is taken of the face and a positive prepared. The defect in the cast is filled with wax or plasteline, and the exact size and shape of the implant are thus obtained. Formerly I used a set of files and saws to prepare the implant; for the last six years, however, for the shaping of ivory, I have used an emery stone driven by a small converter, thus saving time and permitting a more accurate shaping of the implant. The thickness of the skin is deducted from the pattern in order to obtain an implant accurately fitted into the defect (fig. 4).

TABLE 1.—*The Comparative Chemical Analysis of Ivory and Bone*

	Human Bone, per Cent	Ivory, per Cent
Calcium phosphate ($Ca_3[PO_4]_2$)	87.45	82.08
Magnesium phosphate ($Mg_3[PO_4]_2$)	1.57	15.72
Calcium fluoride (CaF_2)	0.35	0.20
Calcium chloride ($CaCl_2$)	0.23	Trace
Calcium carbonate ($CaCO_3$)	10.18	2.04
Ferrous oxide (FeO)	0.10	0.08
	99.88	100.12

TABLE 2.—*Chemical Composition of Vegetable Ivory*

	Per Cent
Gum	6.73
Legumin or vegetable casein	3.80
Vegetable albumin	0.73
Ash	0.61
Water	9.37
Lignin	81.34
	102.58

The obliteration of dead spaces in which blood serum might accumulate will do away with the formation of hematoma which is certain to interfere with the taking of ivory. The fixation of the implant is brought about by its close fitting into the subcutaneous tunnel and later by its encapsulation. A number of years ago I recommended making perforations in the ivory with a fine electrically driven bore, to allow fibrous structures to penetrate the implant and increase its fixation.[18] This procedure is still often used by me.

Asepsis.—The importance of rigorous asepsis in dealing with the ivory substance cannot be overemphasized. Experimentation has proved that ivory is best sterilized by boiling from twelve to fifteen minutes, after which all bacterial flora disappear. Thin sections of a block of ivory sterilized in this way and inserted subcutaneously heal undisturbed; this proves definitely that the inside of ivory is as completely sterilized by boiling as is the outside. Prolonged boiling (for over

one hour) renders ivory slightly brittle, probably through dissolution of its salts.

Insertion of Implant.—One should not attempt to introduce the implant through the endonasal route, because of the great sensitiveness of ivory to infection. The incision for introduction is preferably made along the philtrum or on the lower surface of the nasal tip. A subcutaneous tunnel undermined with long scissors is made and must not extend beyond the size of the ivory support to avoid displacement. The implant should be handled exclusively by appropriate forceps with a close grip and direct contact with the surgeon's hands avoided. The placing of the implant should be done in a way to avoid any pressure of its ends on skin or mucous membrane and thus preclude pressure

Fig. 4 (case 4).—*A*, posttraumatic saddle back deformity resulting from boxing. *B*, condition of patient several years after correction by insertion of an ivory implant through an incision made in the left eyebrow.

necrosis. This represents one of the most frequent causes of failure for the beginner.

The columella incision is closed with fine silk or horsehair sutures, which are removed after two or three days. To assure good immobilization of the implant, a thin lead splint is applied externally. This moderately compressed dressing prevents accumulation of serum and formation of hematoma around the ivory.

Postoperative Reactions and Complications.—During the first few days following the introduction of ivory, there is usually a slight swelling of the nose with redness, which gradually disappears. The reaction is usually the same as that which follows the introduction of cartilage, and, ordinarily, after from seven to ten days, most of the swelling subsides. Occasionally, there is evidence of some exudate, which resorbs in a few days.

In instances in which the exudate is accompanied by redness and swelling that do not subside during the early postoperative period, there is great probability of infection, and the transplant should be removed through a small endonasal incision. Clinical experience has proved that once infected ivory cannot be retained. This is the main disadvantage in the use of ivory as compared with cartilage; the latter, if infected, can be preserved to a great extent when proper drainage and disinfection are carried out. The removal of an ivory implant is, however, without any consequence, being done endonasally after the application of a weak solution of cocaine to the mucous membrane. Later, following the disappearance of the inflammatory process, the same implant can be reinserted through the incision previously made. Infections, from my observation, are always due to faulty technic, the most frequent causes being contamination of the implant before its introduction and hematoma. Both are easily avoided. Pressure necrosis is another late complication which may follow the

TABLE 3.—*End-Results in Sixty-Eight Ivory Implants*

Early removal because of infection	4
Late removal because of pressure necrosis	3
Taking of the implants	61
Total	68

insertion of ivory. Whereas infection manifests itself in the first days following implantation, pressure necrosis may occur months or years after. Usually it is due to excessive length or malposition of the implant which exercises too great a pressure on the skin or nasal mucous membrane at its lower end. This complication can be eliminated by preparing an implant of proper dimension, thus avoiding any pressure on the surrounding covering. A good precaution consists in closing the wound subcutaneously with fine catgut in front of the implant, prior to the suturing of the skin. If necrosis occurs, the ivory implant should be removed by a small endonasal incision. The same implant can be properly shaped and reinserted a few weeks later.

In the course of the last ten years, following the insertion of sixty-eight ivory implants, I have had to remove four shortly after operation because of infection, and three others, respectively, five, nine and fourteen months later because of pressure necrosis. Three of the four infected implants occurred during early attempts when I used to insert the ivory through an endonasal incision. Of the four ivory implants which caused pressure necrosis, three perforated the nasal mucous membrane and one the skin of the lower surface of the nasal tip. In two of these four cases, injury to the nose was directly responsible

for the occurrence; in the remaining two cases, according to the available information, the ivory came out "by itself"; injury as a causative factor could not be excluded with certainty in these cases. There was a possibility, however, that the implants were too long when inserted. In four of the seven cases, the implants were reinserted after proper shaping and, as far as my records show, remained in place.

In four of the seven failures, the implants removed were successfully reinserted.

CONCLUSIONS

The tensile strength of rib cartilage and its lack of absorption make this autoplastic substance popular when a resistant support is required. The natural tendency of rib cartilage to bend, however, is often a drawback to its use in instances in which a long and straight graft is required, especially as it is difficult to foresee when postoperative bending will occur. This tendency is less frequently observed when the cartilaginous graft is of equal thickness throughout; this can be obtained by its resection with a well balanced angular knife. Other contraindications for the use of rib cartilage may be nervous and hypersensitive persons who fear additional scarring of the chest and prolonged hospitalization.

Additional sources of cartilaginous supply are the auricle, alar and lateral nasal cartilages and the septum. The abundance of yellow elastic tissue in the septal cartilage makes for its certain absorption. The auricular and nasal cartilages can be used with advantage in the repair of minor depressions.

True ivory substance morphologically and chemically resembles bone and for this reason does not irritate the human tissues.

Ivory does not absorb and is easy to sterilize and shape.

Extensive clinical experience with true ivory has confirmed biologic assumptions and proved that it is the most satisfactory substitute for cartilage. A strict differentiation of true ivory from vegetable ivory is important, as the pseudo-ivories act as foreign bodies in the tissues.

EFFECTS OF RADIATION ON ALLERGIC NASAL MUCOSA

A FURTHER REPORT

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AND
MAX CUTLER, M.D.
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In October, 1932, we [1] published a preliminary report on the gross and microscopic effects of radiation on the allergic nasal mucous membrane, and noted the clinical results obtained by irradiation of forty patients suffering from hyperesthetic rhinitis. We pointed out that the primary results obtained in this group of patients were uniformly excellent with regard to relief of symptoms, but as sufficient time had not elapsed to be certain of the permanency of these results, further study was needed to interpret properly the therapeutic possibilities suggested by our observations. We also noted that a series of cases of seasonal hay fever was under investigation in order to study the effects of radiation on this disease. Our purpose in this communication is to evaluate this method of treatment of hyperesthetic rhinitis after a period of one year.

A brief review of the morphologic appearance of the nasal mucosa in vasomotor rhinitis recalls that the surface epithelium is the seat of a pronounced hyperplasia, with interrupted areas of desquamated foci. The cilia are absent. Marked edema of the subepithelial tissue, with large dilated tissue spaces filled with serum, is uniformly present, and constitutes one of the characteristic microscopic features of this state. There is marked cellular infiltration of lymphocytes, plasma cells and eosinophils. The ducts and acini of the mucous glands are filled with and dilated by secretions. As the lesions progress, atrophic changes are seen and the mucous glands may disappear. The pathologic changes are confined chiefly to the subepithelial layer. The edema is characteristically transitory. Secondary infection often obscures the true allergic picture.

It should be noted that the initial effect of an adequate radiation on normal nasal mucosa is an erythema. This effect is caused by local

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From the Michael Reese Hospital.
Read before the Chicago Laryngological Society, Dec. 2, 1932.

1. Bernheimer, L. B., and Cutler, Max: Effects of Radiation on Allergic Nasal Mucosa, Arch. Otolaryng. **16**:561 (Oct.) 1932.

circulatory disturbances occurring several days after exposure. The functional changes induced in blood vessels by radiation are well known, since they have been studied directly by the capillaroscope. They consist of erythema and edema caused by a vasodilatation of the capillaries accompanied by diapedesis of leukocytes. According to Gassmann,[2] the lesion is first observed in the nuclei of the endothelium, followed by vacuolization of the cytoplasm. Sometimes the hyperplastic endothelial cells desquamate into the vascular lumen; sometimes they proliferate and obliterate the canal. Vascular lesions are especially

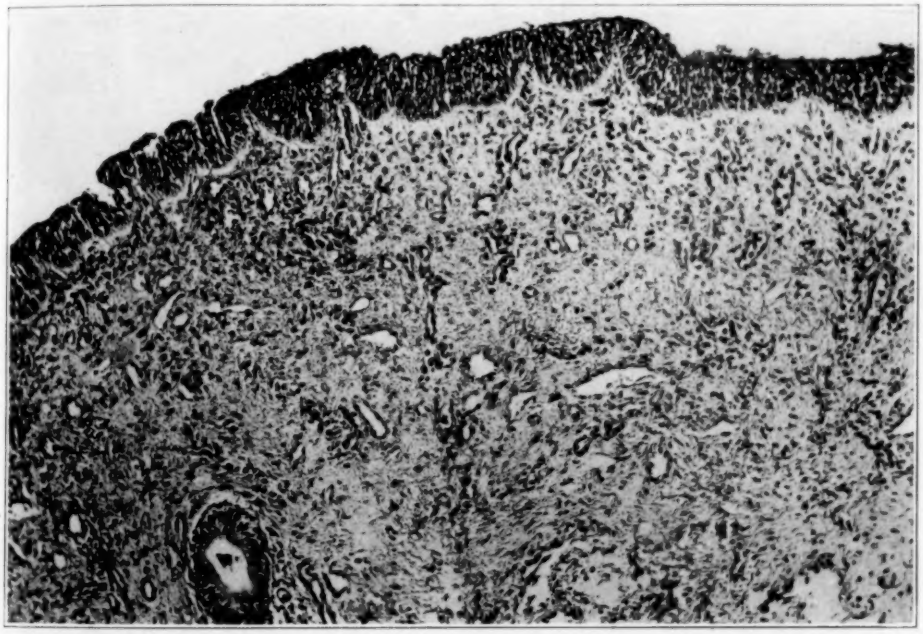

Fig. 1.—Photomicrograph of the nasal mucosa one year after irradiation. The tissue shows a vacuolization of the lining epithelium and interepithelial infiltration of inflammatory cells. There is some edema in the submucosa with a considerable number of fibroblastic cells.

manifest in organs treated by radiation of low filtration. They are particularly noticeable in proximity to radioactive foci introduced interstitially in tissues and also under the influence of diffuse caustic rays (Dobrovolskaia-Zavadskaia [3]).

2. Gassmann, A.: Zur Histologie der Röntgenulcera, Fortschr. a. d. Geb. d. Röntgenstrahlen **2**:199, 1899.

3. Dobrovolskaia-Zavadskaia, N.: Modifications des fibres striées sous l'influence d'irradiations prolongées au moyen de foyers radiferes introduits dans les muscles, J. de radiol. et d'électrol. **8**:69, 1924.

An application of filtered radium to mucous membrane results in a selective cytolytic effect on the germinal layer of the stratified epithelium. This effect occurs during the first days after exposure (Regaud and Nogier [4]). When the dose of radiation is sufficiently strong, there results a true *radio-epithélite* due to a destruction of the germinal layer of epithelium. The destruction and falling off of the surface epithelium occurs about the twelfth day after irradiation. Coutard has shown that under the same technical conditions of irradiation the falling away of the cutaneous epidermis occurs later, i. e., on the fifteenth to twentieth

Fig. 2.—Photomicrograph of the submucous glands one year after irradiation. The mucous glands show no abnormalities. There is a marked infiltration of lymphocytes between the tubules and a new formation of connective tissue. Note the secretion of the epithelial cells and the absence of atrophic changes.

day. Reparation of the lesion takes place between twenty-two and twenty-seven days after irradiation of mucous membranes and about forty-two days after irradiation of cutaneous epidermis.

The effect of radiation on sweat glands and sebaceous glands is a diminution and suppression of their secretions. The mucous glands

4. Regaud, C., and Nogier, T.: Les effects produits sur la peau par les hautes doses de rayons X selectionnes par filtration a travers 3 et 4 millimetres d'aluminium applications a la roentgenotherapie, Arch. d'électric. méd. **21**:49, 1913.

of the nasal mucous membrane are affected in a similar manner. When the irradiation is intense, these glands atrophy and disappear (Rose; Oudin, Barthelemy and Darier [5]). The connective tissue is the most radioresistant and under radiation exposures undergoes a slow process of fibrosis.

The forty persons with hyperesthetic rhinitis who were studied for the preliminary report gave a typical history of spasmodic attacks of sneezing followed by profuse watery discharge and nasal block, the

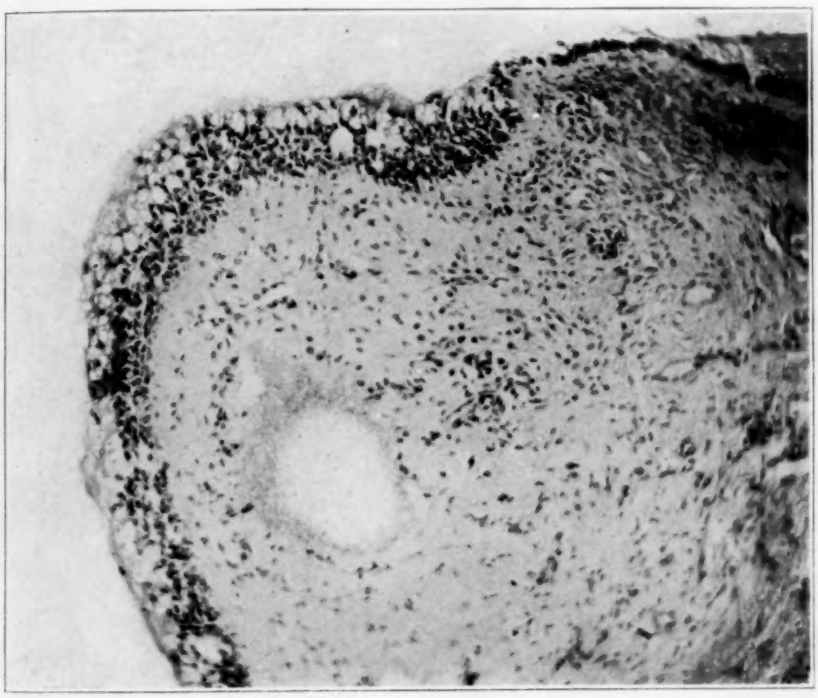

Fig. 3.—Photomicrograph of the nasal mucosa before irradiation, in a patient showing clinical signs of vasomotor rhinitis. Note the presence of distended blood vessels and slight cellular infiltration.

symptoms occurring without relationship to season or climate. Nasal examination in all cases revealed the usual pale waterlogged mucosa found in allergic nasal disease. Eleven of these persons had demonstrated sensitizations to one or more foods or inhalents, showing positive skin reactions to such atopans as orris root, house dust, kapok, chicken feathers, wheat, egg albumin and beef. However, none had responded to allergic therapy. Twenty-nine of the forty persons irradiated had

5. Oudin, Barthelemy and Darier: Accidents cutanés et viscéraux consécutifs à l'emploi des rayons X, France méd. **45**:113, 1898.

previously submitted to some form of intranasal surgical procedures, such as submucous resection, intranasal or sublabial antrotomy, ethmoid or sphenoid exenteration or repeated nasal cautery. The relief obtained from these procedures had been transitory. No patient was irradiated in the presence of polyps or secondary nasal infection, radium being introduced into the nose only after these secondary changes had responded favorably to indicated therapeutic measures.

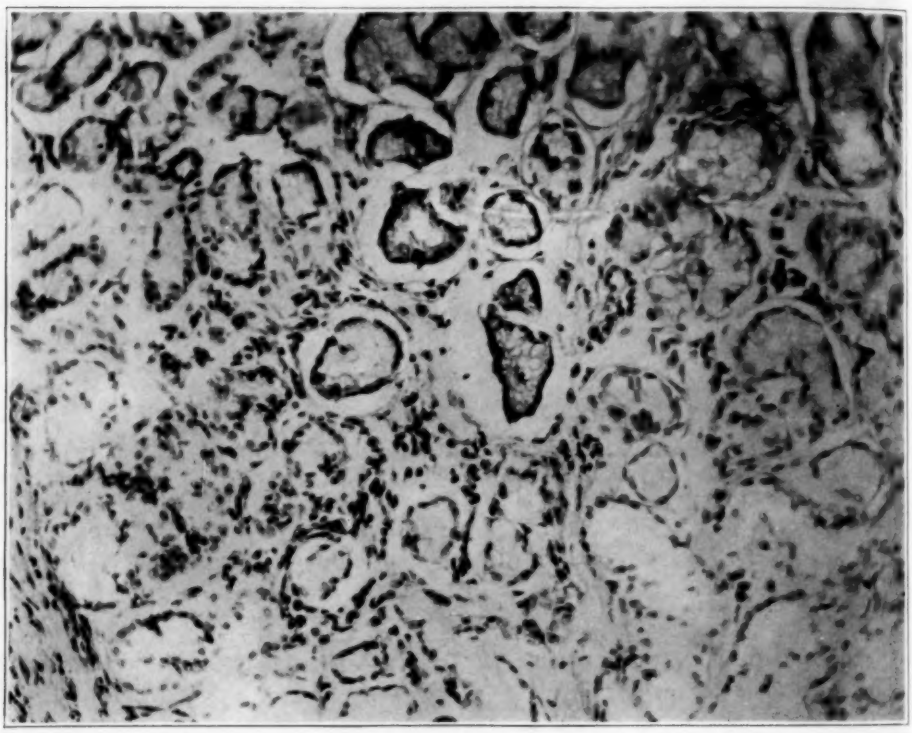

Fig. 4.—Photomicrograph showing nasal mucosa in a case showing clinical signs of vasomotor rhinitis. Note the intense cellular infiltration of the submucosa.

After cocainization of the nasal mucosa, 50 mg. of radium were introduced into each nasal chamber and allowed to remain in place for two hours (total dose 200 mg. hours). The radium element was held in four brass capsules, each capsule containing 25 mg. of radium in silver containers. The total filtration was 0.5 mm. of silver and 1 mm. of brass. One capsule was placed in each middle meatus and one in each inferior meatus. Usually both sides of the nose were irradiated simultaneously.

In our preliminary report we stated that the initial effect of irradiation on the allergic nasal mucosa was an erythema, the congested red nasal membranes causing nasal block. The erythema and block persisted for from three to eight weeks, slowly resolving until the mucosa became normally pink and ample breathing space was established. During this period of erythema and congestion there was a copious mucopurulent discharge, which slowly lessened and finally subsided with the disappearance of the erythema. The sneezing usually stopped immediately following irradiation, so that by the end of the eighth week the irradiated person was symptom-free, the nasal mucous membrane at this time being normally pink; however, in some cases the pale color persisted in spite of the disappearance of the edema.

Observations After One Year of Clinical Results Obtained by Irradiation in Forty Cases of Hyperesthetic Rhinitis

Number of Cases	Sneezing	Nasal Block	Watery Discharge
21 or 52%	Complete relief	Complete relief	Complete relief
8 or 20%	Lessened	Complete relief	Complete relief
3 or 8%	Lessened	Lessened	Complete relief
4 or 10%	Lessened	Lessened	Lessened
4 or 10%	No relief	No relief	No relief

	Number of Cases
Color of nasal mucosa:	
Normal pink color with no waterlogging or block	19
Pale color, but no waterlogging or block	13
Pale color with mild block and mild waterlogging	4
Pale, waterlogged, with nasal block	4
Microscopic section:	
Approached normal appearance	11
Unchanged	1

Microscopic examination of a fragment of allergic nasal mucosa excised eight weeks after irradiation showed pronounced morphologic changes. The subepithelial intercellular edema and dilated tissue spaces which constitute the characteristic microscopic picture in the untreated disease were entirely absent. There was also absence of signs of acute inflammation. Capillary dilatation and leukocytic infiltration had disappeared. The mucous glands were no longer separated by edematous intercellular tissue but were crowded together. Isolated foci of lymphocytes and plasma cells had replaced the previously widespread acute cellular infiltration. The microscopic appearance of the tissue closely approached that of normal mucous membrane.

SUMMARY

Forty persons with hyperesthetic rhinitis were irradiated and the initial observations recorded in an early report. The clinical results observed in this group after one year are shown in the table.

Fifty-two per cent of the patients irradiated were symptom-free after one year; 20 per cent no longer had nasal block or watery discharge, so that while sneezing still persisted in a moderate degree, from the standpoint of the patient's comfort the clinical result was excellent; 8 per cent were moderately comfortable, their breathing space remaining unimpaired, although sneezing and discharge persisted; 10 per cent received but little relief, and 10 per cent were not benefited.

CONCLUSIONS

1. Irradiation of forty patients suffering from vasomotor rhinitis has given satisfactory clinical results in a large percentage of the patients treated. These results have remained constant for a period of one year.

2. The method of radiation described has proved to be safe, no untoward results being observed in any of the persons treated.

3. An analysis of the clinical and histologic effects of radiation of a series of patients with hay fever is under investigation.

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DISCUSSION

DR. MAX CUTLER: The problem which was presented to me for consideration was whether the pathologic state underlying the condition of vasomotor rhinitis is one that may be expected to respond to radium therapy. Actually, the tissue changes associated with vasomotor rhinitis consist of an epithelial hyperplasia of the lining mucous membrane, subepithelial edema and infiltration with lymphocytes and plasma cells and a dilatation of vascular spaces and capillaries. There were adequate reasons for suspecting that these pathologic changes might be influenced favorably by radiation.

The next important consideration was whether the change could be effected safely, particularly as regards the danger of an atrophic rhinitis. The dose that was decided on was a very small one as compared with that used in the treatment of malignant conditions. Furthermore, by employing adequate filtration, the caustic effects of beta radiation are practically eliminated. From all our previous experience there is every reason to believe that the dose employed according to the technic described is safe as regards a complicating atrophic rhinitis.

Microscopic studies of the allergic mucous membrane one year after radiation show a disappearance of the intense cellular infiltration and the submucous edema. The dilated capillaries and tissue spaces are no longer present and the mucous glands show no morphologic signs of atrophy. The microscopic appearance of the tissues is quite normal.

We fully realize that the attempt to benefit this condition by radiation is empiric and that the treatment can in no sense be regarded as specific without an understanding of all the etiologic factors. In this respect, however, it differs in no manner from the radiation treatment of cancer. Careful observation over a long interval will decide the relative merits of this therapeutic procedure, and time alone will establish the safety of the method.

DR. SAMUEL J. PEARLMAN: There are at least two groups of diseases of the nose which are recognized as being closely related to each other. One may be

called seasonal hay fever and the other perennial hay fever. The last-mentioned form goes by other names, such as vasomotor rhinorrhea and allergic rhinitis. The symptoms of both types are too well known to describe here. As the pollens from trees, grasses and weeds produce the seasonal type, the symptoms may often extend over a period as long as seven or eight months. That leaves a group of cases resembling hay fever closely but not dependent on the presence of pollens. The condition is present all year, and is often unaffected by the occurrence of the seasonal variety of hay fever.

The cures that have been suggested for perennial hay fever or vasomotor rhinitis are multiple, and the number of cures offered is pronounced evidence that the condition is poorly understood. It occurs in large numbers of persons and apparently is on the increase. Furthermore, one knows no more about this condition than about atrophic rhinitis and otosclerosis, which have already been discussed.

It is interesting to review some of the supposed causes of this illness. Every one here remembers the time when the sphenopalatine ganglion was regarded as the cause. Only a few years ago many championed the theory of a metabolic disorder. The thyroid, it was said by many, was deficient. Others leaned toward an allergic point of view, for the reason that this disease occurs frequently in families that have other allergic disease. The same patient or his blood relatives may have asthma, hives or eczema. Like asthma, the complaint has a tendency to disappear for a time and recur.

It is curious to review the various types of treatment that have been used. Even at the height of its popularity there was doubt in the minds of some that medication applied to the sphenopalatine ganglion did any good. Many will remember the treatment, frequently successful, consisting of injections of alcohol into the nasal nerves and used by Dr. Otto Stein. For many years Dr. Norval H. Pierce and Dr. Walter Theobald championed the use of trichloracetic acid applied to the turbinates and septal mucosa, often with a satisfactory outcome. There are some instances in which the patient is cured as if by magic if the allergic agent is discovered. There are many ways of discovering the allergic factor. One way is to eliminate the most common offenders. Of these the most important appear to be such things as feathers and cosmetics. The banning of a cosmetic containing orris root may overcome the disease immediately. The same method of elimination may be applied to food factors. Skin testing is frequently unsatisfactory. One may have to use hundreds of possible offending proteins and then be disappointed with the information yielded.

As I understand it, the essayist claims no more for his method than that it is an empiric method. He does not commit himself as to etiology; he says only that radiation in his hands has given complete relief in 50 per cent of the cases for at least one year; other patients are partially improved and some are not at all improved. From the standpoint of empiric treatment I should say that this method is the best yet. I have had no experience with it. It will be interesting to see how long it will be used. It is, furthermore, of interest to know the answer to a question just raised by Dr. Muskat: What happens, after using radium, in those cases in which there is, in addition, itching in the conjunctiva and in the palate and in those that show asthma or a slight trace of asthma? Many patients with vasomotor rhinitis have a little wheezing cough. I should like the essayist to tell what happens to these complaints in addition to those of nasal origin.

DR. IRVING MUSKAT: What percentage of the cured patients have marked nasal allergy with involvement of the sinuses? The amount of radium was no doubt determined as far as the symptoms in the nasal fossa were concerned, but where

there is marked water-logging of the nasal fossa one expects to have polyposis of all the sinuses. If one administers the same dosage, I do not see how one can affect the symptoms arising from the antrum and other sinuses which are quite a distance from the center of radiation.

DR. FRANCIS L. LEDERER: Dr. Pearlman omitted one etiologic factor which is very important in the consideration of hyperesthetic rhinitis. Dr. Bernheimer is the author of a paper, which has been published, in which he enumerated cases that responded to so many different types of therapy that they could not be counted as cures. I refer to a case of mine which he quoted, in which the patient was to have been treated with a calcium preparation intravenously. Dr. Bernheimer had collected a quantity of this preparation and he gave me an ampule of material which was quite different, as we later found out. I used it intravenously, and the patient was cured. One cannot forget that in the treatment of this disease there is the psychic factor. Furthermore, I think that Dr. Bernheimer speaks of the pathology of hyperesthetic rhinitis a little too loosely. I should like to know exactly what he means in his consideration of the disease. He has included such a varied group of cases that it is hard to believe that they are to be considered as purely hyperesthetic cases. It would be impossible to give all the clinicopathologic causes that exist in the literature today grouped under this one head. To my mind the same problem exists as in the cases of many years ago and the various conditions in the nose which were cured, perhaps not by the magic of the material used, but by the personal magnetism of the person who used it. I recall one patient who was examined as a control in a hearing test; following an audiometric measurement he was cured; I also recall a case, previously reported by Dr. Pierce, in a young woman who was affected by roses, which produced paroxysms of sneezing. On one occasion a bunch of roses was brought into the room at a social function which she attended. The patient promptly had an attack of sneezing. The roses, however, were artificial. There are numerous similar cases which might illustrate the neurotic element.

I recently had a newspaper article sent me regarding the radium treatment of sneezing noses. The patient asked me soon afterward why I did not use this method. I asked the editors of the newspaper column why this had appeared so promptly when the work was still untried and unproved, and the response was that Dr. Cutler knew what he was talking about. Dr. Cutler says that he does not know much about the disease nor just how it acts, but he possessed the radium and used it on the general principle that the element causes fibrosis. At the University of Illinois Dr. Fox employed 1 mg. radon seeds, one in the anterior ethmoid and one in the posterior ethmoid area, using four seeds altogether. The report was that his results were not forthcoming. Those patients who were given radium showed definitely the pathologic changes of hyperesthetic rhinitis and asthma. Fox, too, has used radium in many hyperplastic cases, postoperatively. I do not wish to be overly critical, but I think that this is a disease entity which is being included in the class of deafness. Before we know it, we will be forcing these patients to resort to quackery, as in the case of deafness, through the many empiric remedies that have been advised. I think that these cases should always be approached with a good deal of judgment and reservation, as the remedy may become generally known and lead to the indiscriminate insertion of radium into noses. Certainly radium, Dr. Cutler will grant, is not an entirely harmless substance. He says that he does not know on just what basis he used the dosage that he applied, but he placed the radium in the nose and secured just an erythema dose. I think that the entire subject should be taken with much reserve. Certainly these patients should have more than a year's observation before any rash

judgment is given as to the efficacy of such a remedy. The pathology, dosage and possible complications should be studied more carefully.

DR. H. L. POLLOCK: I am much interested in hearing these end-results. All must agree that Dr. Bernheimer did not attempt to go into the etiology. He presented only the result of treatment. We have all had these cases, and I am surprised that any one would criticize the report. Some years ago we had a discussion on similar cases. I reported what I was trying to do in these cases. Some said that they sat back and did nothing. These patients are miserable, and appreciate anything that can be done to alleviate their symptoms.

I think that Dr. Pearlman was taking a shot at me, because I reported the results of injections into the sphenopalatine ganglion, but I did get results in many cases, cure in some and alleviation in many. It took only about two seconds to make the injection into the sphenopalatine ganglion, and it took about nine months or a year for a recurrence to appear.

I am a little skeptical about the final results of this radiation therapy, but some one must be the pioneer in this work, and I think that we should all be grateful to Dr. Bernheimer for presenting it. I think that he uses 200 mg. hours. That may be too much and it may be too little. We can tell only by trying it on the patients. I gave up injections into the sphenopalatine ganglion because it was painful, and the patients would not come back for a second injection. Some of them, however, remain cured to this day. I think that the disorder is due to something which affects the nerves of the sphenopalatine ganglion, and I think that anything that blunts the ends of the nerves is of service. I am going to try this treatment myself in similar cases. I shall be much interested to hear the result of this work in a year or two. Whether there will be some atrophy remains to be seen. One might give an overdose and produce a defect that is worse than the hyperesthetic rhinitis, that is, atrophic rhinitis.

DR. J. GORDON WILSON: Any new method that helps in the treatment of allergic conditions of the nose is to be received sympathetically, especially if, as in this instance, one has the approval of the clinician, the report of the roentgenologist and the evidence presented by the microscopic slides. I confess that I am a little anxious regarding what those slides show. One sees considerable destruction of cavernous sinuses and of glandular structure, with a corresponding increase of connective tissue. What is going to happen later? Will the patient be symptomless so far as vasomotor rhinitis is concerned but have another condition, atrophic rhinitis, in its place? To clear this up I hope that Dr. Bernheimer will tell the outcome a year or longer after this treatment was given

DR. SAMUEL SALINGER: I wish to call attention to the fact that some years ago radium was advocated in the treatment of polyposis of undetermined origin. Polypi which had been removed a number of times and recurred were re-irradiated. I think that the first paper read by Dr. Lyons stated that at that time he used 300 or 400 mg. hours, double the dose used by Dr. Bernheimer and Dr. Cutler. Later he came back and reported using still larger doses. In two cases in which I used radium in sufficient doses to prevent recurrence I got what Dr. Wilson mentioned, namely, an atrophic rhinitis. It takes considerable time for atrophic changes to develop, and one year is not a sufficient length of time to evaluate the final results of the treatment.

DR. HARRY L. HUBER: The etiology of nasal symptoms may be local or constitutional, and treatment should be directed at the etiologic agent. The nasal symptoms are often the end-result, and local applications or removal of tissue gives, at best, only temporary relief.

In the discussion on the pathologic changes in the nasal tissue I was surprised to hear so little said about eosinophils. Recent work indicates that the examination of nasal secretions and tissues gives important information as to the etiology of the nasal symptoms. The presence of many eosinophilic cells is taken as an evidence of allergic etiology and warrants an investigation from the allergic point of view. This investigation should be made thoroughly, as it is often difficult to locate the irritant. In a recent number of *The Journal of the American Medical Association* Dr. Pusey gave a good example of intensive investigation in a case of dermatitis. The same type of investigation is needed to discover the irritant that causes rhinitis in some cases. I have patients who have rhinitis if exposed to certain odors or to changes in temperature. Our investigation of the last-mentioned group shows that 80 per cent are female. They have low blood pressures, low metabolic rates and a slow pulse, and are usually sterile. I think that there is a definite endocrine factor in this group. Dr. Duke of Kansas City believes that many persons with vasomotor rhinitis have a disturbance in the heat-regulating mechanism, and advises exposure of the body to dry heat, followed by ice rubs.

I agree with Dr. Pearlman in his description of the rise and fall of many former methods of treating vasomotor rhinitis, but I think that more credit should be given to allergy. Many cases have been proved to be purely allergic; as the tests are becoming more inclusive and accurate, many more will be correctly diagnosed.

These patients with vasomotor rhinitis are made miserable by their symptoms, and for that reason are willing to do anything that promises to relieve their nasal symptoms. Most of them are overtreated.

I am wondering about the end-results of the treatment with radium. To produce the results described here, there must be a profound change in tissue. In patients with food, epidermal or pollen rhinitis the tissues return to normal when the irritant is removed. If there is a permanent change in tissue after radium, how will persons with such sensitization be affected out of season? How have patients with pollen asthma been affected by radium treatment? I believe that one should be very conservative in the use of radium and that only those who are familiar with its possibilities should use it. Ten years ago, Lyons used it in treating certain types of polypi. Two years ago Hansel advised its use in certain types of vasomotor rhinitis.

DR. L. B. BERNHEIMER: In regard to the treatment of patients with hyperesthetic rhinitis complicated by pulmonary or conjunctival symptoms, no persons with associated asthma were irradiated, but in a few in whom conjunctival symptoms were present the ocular irritation improved with the improvement of the nasal condition. One patient with chronic asthma, who failed to respond to any form of therapy and who did not have hyperesthetic rhinitis, was irradiated with the radium bomb, short exposures to the chest being employed. The results were nil.

In regard to irradiating patients with hyperesthetic rhinitis complicated by suppurative or polypoidal sinus disease: In the original paper on the subject of irradiation of patients with hyperesthetic rhinitis, there is a statement that no patient was irradiated until the polyps or suppurative sinus disease was eradicated by indicated operation or local treatment. No patient with hyperesthetic rhinitis was irradiated even in the presence of ordinary nasal infection, the infection first being cleared up before radium was introduced into the nose.

In regard to an allergic pathologic process in the sinuses, associated with allergic pathologic changes in the nose, radium was employed in the nasal cham-

bers, penetrating the sinuses, the mucous membrane of the sinus receiving almost as concentrated radiation as the mucous membrane in the nose itself, so that if an allergic pathologic process was present in the sinus, it responded in the same manner as the mucous membrane of the vaults of the nose.

Replying to Dr. Wilson, the youngest patient treated was 17 years old.

Dr. Lederer seems to doubt my ability to diagnose hyperesthetic rhinitis. I do not believe that there is anything particularly difficult in making this diagnosis. If he will read the preliminary report on the subject of the effects of radiation on the allergic mucosa, he will find a full description, both gross and microscopic, of the pathologic changes encountered. I am sure that there could be no confusion between hyperplastic rhinitis and hyperesthetic rhinitis, and I am sure that no mistake was made in the diagnosis.

Dr. Huber does not have quite the right conception of the value of radiation in the treatment of allergic nasal disease. We fully realize that the problems underlying the mechanism of hyperesthetic rhinitis are not simple local entities in the nose itself, and we have made no statement to this effect. We fully realize that all allergic manifestations undoubtedly have constitutional factors; some of these are understood, but many of the most important cases remain unexplained.

In view of this understanding, the treatment of allergic nasal disease by radium is not offered as a substitute for known and recognized allergic procedures, but as a useful adjunct in the many cases in which allergic treatment fails to benefit the patient. Certainly if an obvious sensitization is responsible for hyperesthetic rhinitis, such as sensitization to orris root, the simple procedure is to remove orris root from the powder used by the patient, but these obvious sensitizations are rare. The usual experience is to find sensitization to a group of atopens with which the patient has had no contact, e. g., sensitization to lobster in a person who cannot even afford a flank steak.

I repeat that we do not pretend that this treatment is etiologic, but we do say that it is symptomatic and most satisfactory, and it is presented as a safe method of relieving symptoms in hyperesthetic rhinitis. We have reported the results after a period of one year. I shall be glad to report results later. I hope that others will try the described treatment, using the dosage that we recommend, which I am sure is safe. I shall be happy if they will communicate with me to tell what results they have obtained.

PROCAINE CRYSTALS AS A LOCAL ANESTHETIC FOR INTRANASAL SURGERY

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In preparing a paper on the use of procaine crystals or mud as an anesthetic for intranasal surgical procedures, I feel as though an apology is in order. With that mental attitude, I decided to satisfy my curiosity by determining just how much had been written on the subject. Consequently, I was much surprised to find that the literature of the past fifteen years made practically no mention of the surface application of procaine mud. It is for this reason that I present this paper.

Bowers [1] of New York has been using procaine crystals as an anesthetic in the nose for several years with great success. The general consensus, as obtained from the literature, seems to be that procaine is to be used in solution by the infiltration method. Thomson,[2] however, in his book, stated that procaine can be applied to the mucous membranes in solution of the same strength as indicated in cocaine.

The disadvantages of cocaine are familiar to all, namely, its toxicity, expense, habit-forming tendencies, and last but not least, the peculiar susceptibility which some persons have for the drug. Cocaine has been the local anesthetic of choice for many years. Procaine is a more recent drug, and having much the same name often leads one into thinking that its chemical structure and actions are similar to those of cocaine. The two drugs are entirely different. Smith [3] stated that many members of the medical profession are yet under the impression that procaine is closely related to cocaine, which is not the case. It is true that both procaine and cocaine produce local anesthesia, but these two compounds are entirely different, each one possessing an entirely different arrangement in the chemical make-up of the molecules. Cocaine is an alkaloid obtained from the cocoa plant, while procaine is a synthetic preparation made from chemicals.

In comparing procaine with cocaine, one finds the former to be much less toxic, not habit-forming, and relatively inexpensive. Cocaine crystals cost about $14 an ounce, while the same quantity of procaine crystals costs $3. Procaine does not come under the narcotic regulation.

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1. Bowers, Wesley C.: Personal communication.
2. Thomson, St. Clair: Diseases of Nose and Throat, ed. 3, London, Cassell & Company, 1926, p. 84.
3. Smith, A. E.: Block Anesthesia and Allied Subjects, St. Louis, C. V. Mosby Company, 1921, p. 239.

My experience with the use of procaine began three years ago while on the house staff of the ear, nose and throat service of Bellevue Hospital. Previous to that time cocaine in the form of crystals or solution had been used exclusively for surface application in operations within the nose. Since that time, I am confident that from a third to a half of the intranasal operations performed by the house staff have been done with procaine. The total number of operations would be between five and six hundred, consisting of submucous resections, turbinectomies, removal of polypi, exenteration of ethmoids and sphenoids and window operations on antrums. My personal records show that I have performed over one hundred and fifty intranasal operations, and the anesthesia proved to be most satisfactory, both to the patient and to the surgeon. In the entire series, I did not observe a single reaction. I doubt if that can be said of cocaine.

Several years ago the Therapeutic Research Committee of the Council on Pharmacy and Chemistry, acting under the authority of the American Medical Association, appointed a special committee to collect and study information on the occurrence of accidents from the use of local anesthetics. The chairman of this committee, Emil Mayer,[4] reported forty-three deaths, twenty-six of which were due to cocaine and two to procaine. Of these two, one could not be classified as having been caused be procaine, because both cocaine and procaine had been used as the anesthetizing agent. In the one remaining case death was classified as being caused by procaine, but even here the application had been by infiltration and not on the surface. I have not been able to find a single instance in which the surface application of procaine has caused death.

Another advantage of procaine as compared with cocaine is the ease and safety with which it can be used on children. These young patients are particularly sensitive to cocaine, and personally I am reluctant to use it at all on them. I have found that antrum irrigations can be done satisfactorily in children by using procaine as the anesthetic. This accomplishment, in my opinion, is sufficient proof that procaine does all that any local anesthetic is expected to do. Sollmann [5] stated that after a series of experiments he found cocaine to be most efficient and procaine relatively inefficient on surface application. I will admit that to produce the same degree of anesthesia one would have to use a little more of procaine than cocaine, but when the margin of

4. Mayer, Emil: The Toxic Effects Following the Use of Local Anesthetics, J. A. M. A. **82**:876 (March 15) 1924.

5. Sollmann, Torald: The Comparative Efficiency of Local Anesthetics, J. A. M. A. **70**:216 (Jan. 26) 1924.

safety of procaine over cocaine is considered, this becomes a trivial matter. Clinically, I find that procaine is just as efficient so far as the duration and depth of anesthesia is concerned.

The technic is simple and consists of placing a small amount of procaine crystals in a water glass or any suitable receptacle and moistening a cotton wound applicator with epinephrine, which is then dipped into the crystals; the procaine that clings to the cotton applicator is rubbed on the surface to be operated on. This is repeated until complete anesthesia is obtained, care being taken to block the sphenopalatine ganglion as is customary when cocaine is used.

CONCLUSIONS

I suggest the use of procaine crystals or mud instead of cocaine crystals or solutions, because they are, (1) less toxic, (2) not habit-forming, (3) practically as efficient as cocaine and (4) less expensive.

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ABSCESS OF THE BRAIN FOLLOWING MILD TRANSITORY OTITIS MEDIA

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AND
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The successful treatment of abscess of the brain depends on timely diagnosis as well as on proper surgical procedure. Timely diagnosis means diagnosis often in the absence of localizing signs and in the presence of comparatively vague signs of increased intracranial pressure. If diagnosis of an abscess of the brain is deferred until definite paralysis, aphasia or other localizing signs develop, with choked disk, many patients will be moribund and perhaps die before surgical drainage can be established. It is fairly well recognized that aural suppuration is the most common single cause of abscess of the brain, so that patients with such cases are usually referred to the otologist early in the disease. Moreover, the patient himself, being conscious of the discharge from his ear, is apt to connect the cerebral symptoms with the otitic trouble and consult an otologist.

That an abscess of the brain can develop following very mild, transitory otitis media is not well known. The first symptoms of intracranial involvement may appear one or several weeks after mild otitis media has entirely subsided. Nearly all patients with such cases are seen first by the general practitioner or the pediatrician, since the patient does not connect his present symptoms with his entirely healed otitic trouble.

A review of the literature revealed the apparent rarity of this condition. Macewen,[1] in his classic work on intracranial suppuration published in 1893, stated that it is highly improbable that an abscess of the brain can form from otitis media and remain while the disease of the middle ear becomes cured, though he mentioned one such case from the literature. Paul Henius,[2] in a careful review of the literature in 1923, was able to find only seventeen cases of abscess of the brain

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Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Jan. 6, 1933.

1. Macewen, William: Pyogenic Infective Diseases of the Brain and Spinal Cord, Meningitis, Abscess of the Brain, Infective Sinus Thrombosis, Glasgow, Maclehose & Sons, 1893.

2. Henius, Paul: Acta oto-laryng. **5**:195, 1923.

following healed otitis media reported in the past fifty years. However, in most of these cases the otitis media was severe and associated with operation on the mastoid, so that in only eight cases did the abscess develop after mild, transitory otitis media. Schmiegelow [3] cited an additional case of this sort in 1929. Eagleton,[4] in his book on abscess of the brain, mentioned one such case.

In the service of one of us (Dr. Faunce) at the Massachusetts Eye and Ear Infirmary, during the first three months of 1932, six patients with abscess of the brain were seen by us and by our chief, Dr. D. Harold Walker. Five of the patients recovered following surgical drainage. In two cases abscess developed subsequent to an operation on the mastoid for severe acute otitis media. An abscess developed in one case of chronic suppurative otitis media. In three patients, or half the number observed during this period, abscesses developed following very mild acute otitis media which completely healed before the onset of the first symptoms of intracranial involvement. In these cases, a history of antecedent otitis media was obtained only by specific questioning. The diagnosis was made in two of these three cases by the close cooperation between the medical services of the Massachusetts General Hospital, where the patients were admitted, and the otitic service of the Massachusetts Eye and Ear Infirmary. In all three cases the tympanic membrane was intact with no signs of inflammation. The mastoid cells were free from suppuration. The bone under the dura was normal, and the dura itself was normal and not adherent to the surface of the bone. Two of the three patients recovered following operation. The third one died, and autopsy revealed the probable pathway of infection from the middle ear to the brain.

REPORT OF CASES

CASE 1.—W. V., a boy, aged 9, was taken to the emergency ward of the Massachusetts General Hospital on Jan. 8, 1932, because of severe headache. Three weeks before admission he had had a cold in the head with fever and pain in the left ear for which paracentesis was done. There was a discharge from the ear for four days, following which the patient seemed well for about ten days. Eight days before admission he had had generalized convulsions which lasted two hours. Since then he had complained of intense pain behind the left eye at intervals, with frontal headache.

Because of the history of otitic trouble, the pediatrician called an otologist in consultation; he found both drum membranes intact; the left tympanic membrane, however, appeared thick and pale. There was no edema or tenderness of the mastoid. Neurologic examination gave negative results except for slight rigidity

3. Schmiegelow, E.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:273 (Aug.) 1929.

4. Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operative Technic, New York, The Macmillan Company, 1922.

of the neck. The fundi showed early choking of both disks. Lumbar puncture revealed a pressure of 300 mm. of water, 5 cells (lymphocytes) and a positive globulin reaction (ammonium sulphate). A diagnosis of probable abscess of the left side in the temporal lobe was made, and the patient was admitted to the Massachusetts Eye and Ear Infirmary.

The next morning the patient was stuporous, occasionally vomited and had a pulse rate at times as slow as 65. A preliminary mastoidectomy was performed on the left side and showed a little exudate in the cells around the antrum; there was no breaking down of cell partitions or other pathologic changes. The lateral sinus appeared normal. A large area of the dura of the temporal lobe was exposed. This was tense but otherwise normal. With coagulation diathermy a circular area 4 cm. in diameter was outlined on the dura to obliterate the meningeal spaces, thus minimizing the danger of operative meningitis. Three days later exploration made at a point within the diathermatized area revealed a large abscess of the temporal lobe 1.5 cm. from the surface, from which an ounce of odorless, creamy pus was drained. A pure culture of Pneumococcus, type III, was obtained. A wire basket drain was inserted; the patient rapidly improved and was discharged seven weeks later, the wound being completely healed.

Case 2.—E. J., a girl, aged 13, was admitted to the Massachusetts Eye and Ear Infirmary on Jan. 27, 1932. Four weeks before admission pain had developed in the left ear; it was relieved by paracentesis, followed by suppuration which lasted four or five days. The ear then became dry, and she seemed entirely well until two weeks before admission, when she began to have occipital headaches with occasional vomiting. These symptoms increased, and a week before admission she began to hold her head toward the right shoulder, any motion of the head from this position causing vomiting.

The ears were normal except for a slightly scarred and retracted left drum membrane. The fundi were normal. Neurologic examination showed definite adiadokokinesis of the left hand with spontaneous past pointing to the left. Lumbar puncture revealed normal pressure, 2 cells and a faintly positive globulin reaction. Caloric tests showed active labyrinths on both sides but abnormal responses from the left static labyrinth. A diagnosis of probable left cerebellar abscess was made.

A preliminary mastoidectomy performed on the left side showed only a little serum in the cells around the antrum with no destruction of the cells. The lateral sinus appeared normal. The cerebellar dura was exposed and appeared normal. Diathermy was applied to the dura as in case 1. A week later exploration of the brain carried out within the diathermatized area, as in case 1, revealed a fairly large left cerebellar abscess 4 cm. from the surface, containing several drachms of odorless, greenish mucopus. Examination of the pus showed a pure culture of Pneumococcus, type III. The abscess was drained by a small rubber tube. The patient improved steadily and was discharged well two months after admission.

Case 3.—A. B., a boy, aged 10, was admitted to the Massachusetts General Hospital on March 3, 1932, complaining of headache. Four weeks previously he had had bilateral earache followed by a bilateral discharge lasting two or three days.

He seemed well for two weeks; then headache began on the right side. The pulse was slowed at times. Neurologic examination gave normal results. The fundi showed bilateral choked disks of 2 diopters. Lumbar puncture showed an initial pressure of 350 mm. of water, 36 cells and a positive globulin reaction.

An otologist was called in consultation because of the history of past otitic trouble. He found the right drum membrane somewhat thickened and slightly reddened along the handle of the malleus, but dry and intact. The left drum membrane was normal. A diagnosis of probable abscess of the right temporal lobe was made, and the patient was transferred to the Massachusetts Eye and Ear Infirmary.

A preliminary mastoidectomy performed on the right side showed the cells to be intact with some granulations in the zygomatic region. The lateral sinus was not exposed. The dura of the temporal lobe was widely exposed and appeared tense but otherwise normal. Diathermy was applied as in the other cases. The following day a slight facial weakness was observed on the left, the only neurologic

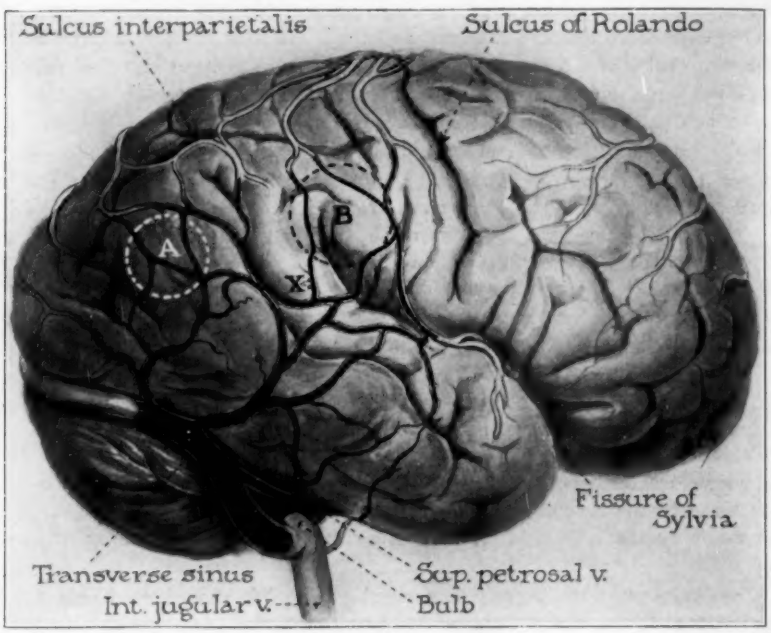

Fig. 1.—*A* and *B* indicate the sites of subcortical abscesses of the brain. *X* indicates the area from which tissue was taken for the microscopic section shown in figure 2. The thrombosed portion of the lateral sinus and the thrombosed pial veins are dark; the normal veins are light.

finding present in this case. Exploration of the brain through the diathermatized area failed to locate an abscess. The slow pulse, increased choking of the disks and severe headache persisted, and a second exploration was carried out four days later; no abscess could be demonstrated. That night the patient died suddenly.

Autopsy revealed two abscesses, one in the right temporal lobe superiorly, nearly in the parietal lobe, and the other posteriorly nearly in the occipital lobe of the same side. The right lateral and superior petrosal sinuses were occluded by a firm, organized, white thrombus. The pial vessels running up over the surface of the right temporal lobe were greatly thickened, yellowish and bloodless (fig. 1). Each abscess was about the size of a golf ball, with a firm capsule, and

was situated in the white substance beneath the cortex. There was no local or generalized meningitis. The dura was normal except where it had been exposed at operation and diathermatized. A pure culture of Pneumococcus, type III, was obtained from the abscess.

Microscopic sections prepared in Dr. Mosher's laboratory showed a well organized thrombus of the sigmoid and superior petrosal sinus. The pial veins over the surface of the temporal lobe were occluded by a well organized thrombus, but with a marked perivascular accumulation of polymorphonuclear leukocytes.

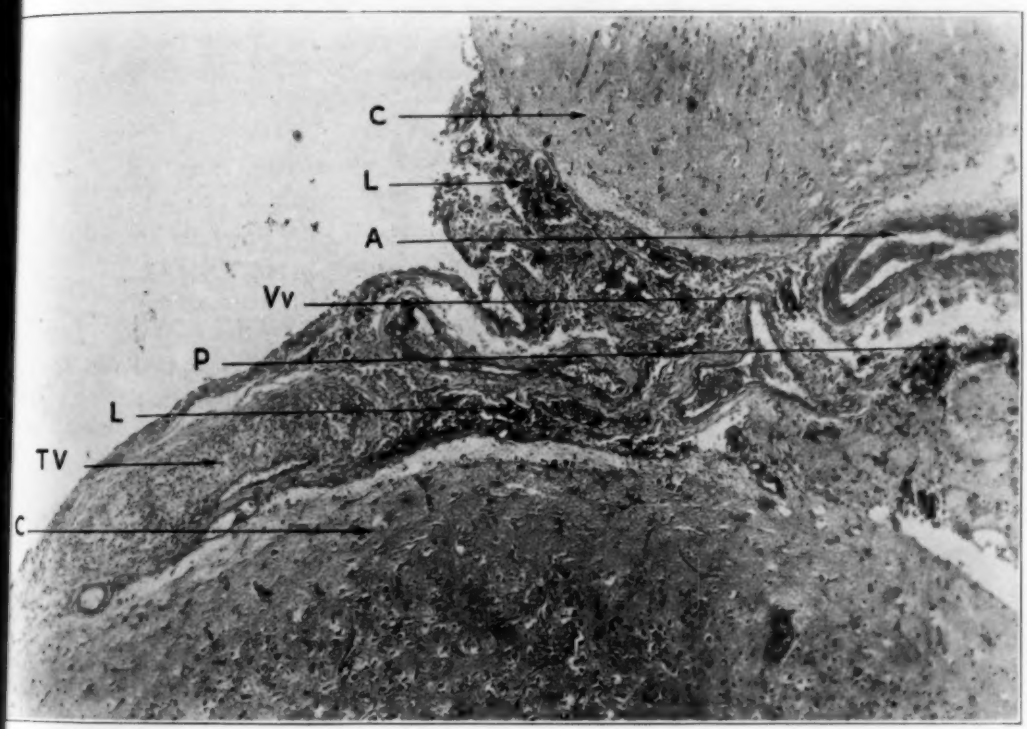

Fig. 2.—*C* indicates the cortex of the temporal lobe; *L*, perivascular infiltration of lymphocytes; *A*, a normal pial artery; *Vv*, normal pial veins; *P*, perivascular infiltration of polymorphonuclear leukocytes; *TV*, a thrombosed pial vein.

The pathway of infection from the middle ear to the two distant abscesses of the brain was apparently by a phlebitis and thrombosis of the small veins of the middle ear leading to the lateral and superior petrosal sinuses and thence, in a retrograde manner, to the pial veins draining the temporal lobe. At two separate points the process broke down into abscess formation in the white substance of the brain where the circulation is poorer than in the cortex. Except for these points of low resistance the infection remained localized and latent in the veins (fig. 2).

Since the lateral sinus was found to be normal in the first two cases, the infection probably passed directly through the small venules of the middle ear which penetrate the thin bone of the tegmen and pierce the dura to connect with the vessels of the pia. Such a venous pathway of infection explains how a mild otitis media can produce an abscess of the brain without destruction of bone in the mastoid, an extradural abscess or localized meningitis. This mode of infection has been suggested by Wittmaack.[5] Eagleton mentioned its possibility in his book on abscess of the brain, while Turner and Reynolds[6] have recently demonstrated the importance of venous thrombosis in the spread of infection from the nasal sinuses to the intracranial structures. The presence of a pure culture of pneumococcus, type III (Streptococcus mucosus-capsulatus), in all three cases should be noted.

COMMENT

We are inclined to believe that the incidence of abscess of the brain following mild, transitory otitis media is greater than is generally appreciated; the history of otitis media is disregarded or not obtained, the patient dies before a diagnosis is made, or autopsy reveals an abscess of the brain in the presence of normal temporal bones, the final diagnosis being abscess of the brain of unknown origin. The fact that we have observed three such cases in the course of three months, half the total number of abscesses seen in this period, would indicate that the condition is probably not rare. Any case in which vague or definite signs of intracranial disease develop and in which a history of antecedent otitis media can be obtained should be considered a possible case of abscess of the brain.

CONCLUSIONS

1. Abscess of the brain may follow mild, transitory otitis media.
2. The mode of infection in these cases is probably by a thrombosis of the venules of the middle ear extending through the dura to the brain.

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5. Wittmaack: Ueber die Beziehungen der pneumatischen Störungen zur Entwicklung endokranieller Komplikationen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **97**:7, 1915.

6. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases, Edinburgh, Oliver & Boyd, 1931.

ELECTRIC THERMOSCOPE AS AN AID IN THE DIAGNOSIS OF ACUTE MASTOIDITIS

PRELIMINARY REPORT

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The majority of cases of acute mastoiditis fortunately present no difficulty in diagnosis. There are, however, so-called borderline or atypical cases in which the diagnosis of acute mastoiditis is so uncertain that the question of surgical intervention is a perplexing problem. Often in these cases delay in an operative procedure may mean disaster. Occasionally, one sees otitic infections which show no definite clinical signs or symptoms of surgical mastoiditis, yet one feels that something is amiss. If there were some test or sign which would give one an inkling as to whether or not the infection of the ear was resolving, one would be relieved of considerable worry. Some of the patients feel so well that they refuse operation even if the surgeon is inclined to believe that the mastoid bone should be operated on. All in all, it is a most harassing problem to the otologist.

In acute mastoiditis, the constitutional temperature is of no real significance. Particularly in atypical cases, the patient may have a very low temperature, or it may even be normal. In spite of this, progressive pathologic changes may go on in the mastoid bone. On the other hand, I find that irrespective of the constitutional temperature the local temperature of the drum of the involved ear as compared with the temperature of the normal drum always gives information which is constant and reliable. Thermometry has shown that the temperature of the mouth is not quite 1 C. (1.8 F.) higher than the temperature of the normal tympanum. In acute otitic infections, the temperature of the involved tympanum is from 0.5 to 2 C. (0.9 to 3.6 F.) higher than that of the normal drum, or is equal to, or up to 1.5 C. higher than, the oral temperature. If the involvement of the ear begins to resolve, the temperature of the ear falls and approaches the temperature of the normal drum or drops below the oral temperature. If the elevated temperature of the involved ear persists, an operative case of mastoiditis eventually develops.

Dr. B. M. Becker[1] published an article in the ARCHIVES OF OTO-LARYNGOLOGY in February, 1930, in which he described the use of the

1. Becker, B. M.: Aural Thermometry as a Diagnostic Aid in Otology, Arch. Otolaryng. **11**:205 (Feb.) 1930.

thermometer to compare the aural temperature and the oral temperature. I have found certain technical difficulties in using his method. In the first place, one cannot be certain that the thermometer bulb is properly placed against the tympanum, and consequently the temperature of the external canal may be obtained. Then again, this method cannot be used in very narrow external canals or in small children. At times it is a tedious procedure to the otologist and a painful one to the patient. Furthermore, it is not the actual temperature that one is interested in, but the difference in temperature between the involved tympanum and the normal one or between the involved tympanum and the mouth. It therefore is not thermometry that gives the diagnostic clue, but thermoscopy, which means an indication of a difference in temperature between two points. The logical instrument to use is a thermoscope, which is a differential thermometer indicating slight differences of temperature without registering or recording them.

THE PHYSICS OF THERMO-ELECTRICITY

In 1921, Seebeck, of Berlin, discovered that in a circuit made of two different metals, if one junction is hotter than the other, there is an electromotive force which causes an electric current. This electromotive force is very small compared to ordinary battery cells; consequently, to obtain much current, the circuit must have very low resistance. In a copper-iron circuit, when one junction is at 100 C. and the other at 0 C., the electromotive force is about 0.001 volt, and causes an electric current from copper to iron at the hot junction, and from iron to copper at the cold one. The introduction of another metal does not make any difference, provided the two junctions of the new metal are at the same temperature.

The thermo-electric method of measuring temperature is based on this principle. For ordinary or low temperatures a copper-iron or an iron-German silver junction may be used. For high temperatures, however, the junction of pure platinum and platinum-rhodium alloy is employed. If a sensitive galvanometer is introduced in the circuit, it becomes a delicate means of measuring differential temperatures.

ELECTRIC THERMOSCOPE

The instrument that I use is known as the thermocouple. I call it the electric thermoscope. It is small and portable, and the use of this type is extremely simple, so that a test can be made in a few minutes. The slightest difference in temperature between two points is indicated by a galvanometer graduated in degrees centigrade, and so constructed that a plus or minus reading of the two points tested can be made. The tip of one terminal is known as the "hot point," and the tip of

the other I call the "testing point." The testing wires are so fine that they may be inserted directly against the tympanum in the narrowest canal or can be used in the most sensitive patient. By the use of this instrument, one can be certain of actually testing the differential temperature of the drum as compared with that of another point. If both points tested are of the same temperature, the reading of the galvanometer will be 0. If the hot point is placed on the warmer part, the galvanometer will indicate a minus reading, and vice versa. In testing two normal drums, a reading of 0 will be obtained. The hot point held in the mouth against the tongue and the testing point placed in contact with a normal drum will give a reading of about minus 1 C.

DIAGNOSTIC AND PROGNOSTIC VALUE OF ELECTRIC THERMOSCOPY

I have observed that in acute infections of the middle ear when mastoiditis is suspected, the thermoscopic reading as compared with that of the normal tympanum varied from plus 0.5 to plus 2 C. In using a normal drum as a means of comparison, the hot point is placed in the normal ear and the testing point is placed in the involved ear. In the event that both ears are involved, I use the mouth as my standard of comparison; the hot point is then placed in the mouth and the testing point is inserted in the ear that I wish to test. Under these circumstances the reading varies from 0 to plus 1.5 C. (2.7 F.). If repeated tests show a continued or persistent plus reading, my experience has shown me that the infection is not resolving and that the mastoid bone is undergoing pathologic changes. On the other hand, if such readings of the instrument indicate that the differential temperature is dropping and becoming equalized, one may be sure that the infection of the ear is resolving. This simple test has relieved me of considerable anxiety in borderline cases of acute mastoiditis, and up to the present time has not failed me. I have utilized this test for the past two years in many cases, and I am now beginning to use it as a routine procedure in all acute otitic infections. Often one can almost prognosticate the course and outcome of an acute infection of the middle ear.

REPORT OF CASES

CASE 1.—R. L., a man, aged 39, was seen by me on Jan. 22, 1931. He complained of pain and a discharge from the left ear for eight days. Examination of the right ear gave normal results. The left drum was full and markedly congested. A moderate amount of seropurulent discharge was seen to pulsate through a very small perforation in the posterior inferior quadrant. There was slight tenderness over the tip of the mastoid bone, and marked deafness was noted. The rectal temperature was 99.6 F. The thermoscopic reading of the left drum as compared with that of the right was plus 1.5 C. Since drainage appeared to be poor, I performed a myringotomy, and a culture of the discharge showed a few

colonies of pneumococci and Bacillus mucosus-capsulatus and a moderate growth of Staphylococcus aureus. On January 24, the deafness and pulsations in the ear persisted, but the tenderness over the tip had disappeared. The thermoscopic reading was plus 1.5 C. Two days later the patient felt much better, but the thermoscopic reading was still plus 1.5 C. A roentgenogram of the mastoid bone taken on January 28 by Dr. L. W. Cole was reported as showing in part: "The outlines of the cell walls are indistinct with destruction of cells in the posterior aspect." The patient felt so well that I had difficulty in convincing him that he had a serious condition. However, since the thermoscopic readings persistently stayed up, varying from plus 1 to plus 1.5 C., I insisted that he be operated on. A mastoidectomy was performed and revealed extensive pathologic changes. He then made an uneventful recovery.

Case 2.—I. M., a boy, aged 9 years, was seen by me at my office on May 13, 1932. He complained of pain in his right ear. Examination of his left ear revealed a normal drum. His right ear was bulging and reddened, with a loss of all landmarks. The rectal temperature was 103 F.; the thermoscopic reading, plus 2 C. I performed a myringotomy, which was followed by a profuse serosanguineous discharge. On May 15, the thermoscopic reading was plus 1 C.; on May 17, plus 0.5 C. From then on the patient made an uneventful recovery.

Case 3.—J. G., a man, aged 50, was seen by me at my office on Oct. 8, 1932. He complained of pain and discharge from the right ear for six days following an attack of influenza. Examination of the left ear showed it to be normal. The right drum was reddened, with a perforation of the antero-inferior part of the drum. A thin mucopurulent discharge was seen pulsating through the perforation. He also complained of severe headaches, marked loss of hearing and inability to sleep. The tip of the mastoid bone on that side was very tender, and tenderness was also elicited over the antrum. The rectal temperature was 100.6 F. The thermoscopic reading of the right ear as compared with that of the normal drum was plus 1 C. On October 10, the patient still complained of severe headaches, pulsations and marked deafness. The thermoscope indicated plus 1 C. Four days later the symptoms persisted, and the thermoscopic reading was plus 1 C. These findings were the same for the next five days, and I was prepared to admit him to the hospital. However, on October 19, the thermoscopic reading dropped to plus 0.25 C. Here was my clue, and I decided to wait one more day. On October 20, the headaches improved, the pulsations diminished and the hearing improved. Two days later, the headaches had entirely disappeared, with abatement of all other symptoms. The thermoscopic reading dropped to 0. From then on the patient made an uneventful recovery, and was discharged on October 28.

Case 4.—J. J., a man, aged 37, was seen by me at my office on Jan. 6, 1933. Five days before, he had an attack of influenza, followed by pain in the right ear and spontaneous rupture of the drum. Since then he had complained of severe headaches, deafness, pain and discharge from the right ear. Examination of the left ear revealed it to be normal. The right ear showed a congested, full drum and a pulsating discharge through an inadequate perforation in the inferior portion of the tympanum. The rectal temperature was 99.4 F.; the thermoscopic reading, plus 1.5 C. Because of the poor drainage, I performed a myringotomy, with some relief of the symptoms. On January 11, although the headaches, pain and pulsations abated, the fulness of the drum and the deafness persisted. The thermoscopic reading was plus 1 C. Examination on January 13 showed no change in the clinical picture, but the thermoscopic reading rose to 1.5 C. The condition remained unchanged until January 18, and I advised hospitalization. On January 20, although the clinical picture remained unchanged, the thermoscopic reading dropped to plus

0.5 C. Here again was my diagnostic clue, and I decided to wait twenty-four hours. On January 21, all the symptoms were greatly improved, and the clinical picture began to change. The thermoscopic reading was plus 0.25 C. I was now certain that the infection of the ear was resolving. On January 23, the headaches and pain had entirely disappeared, and hearing was quickly being restored. The thermoscopic reading became 0. From then on, the patient made an uneventful recovery.

CONCLUSIONS

1. Electric thermoscopy is a reliable diagnostic aid in cases of atypical acute mastoiditis.

2. It is more accurate and a much more simple procedure than aural thermometry.

3. Thermoscopy is applicable under conditions in which aural thermometry is impossible.

4. By the use of the electric thermoscope, a differential temperature reading of two points tested is immediately obtained, irrespective of the constitutional temperature.

5. The electric thermoscope is ideal for bedside purposes, as it is small and portable.

6. The principle of thermoscopy can be easily applied and utilized in sinus infections.

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EFFECT OF DESTRUCTION OF ONE LABYRINTH ON REACTIONS TO ROTATION

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The pharmacology of the vestibular apparatus has been the subject of our investigation for some time.[1] The work up to the present has been done chiefly on normal dogs. In our last article [2] the only recorded operative procedure consisted of sectioning the posterior columns of one side of the spinal cord in the occipital region. It is generally considered, anatomically, that the vestibular apparatus is divided into two equal asymmetrical parts. Since one side is often destroyed by injury or disease, it was deemed of interest to study the reactions to rotation after the destruction of one labyrinth. It was thought, also, that this condition might reveal to advantage the action of certain drugs.

EXPERIMENTAL WORK

Dogs were used as subjects. The animals were anesthetized for operation. An incision was made through the skin, back of the mastoid region. The muscular attachments below the nuchal ridge and back of the postmastoid ridge were separated over an area of about 1 sq. cm. A large ear speculum was inserted to retract the soft parts. Through this speculum a large dental drill was passed to the apex of the angle of the two ridges. The drill was advanced through the bone toward the center of the lower edge of the opposite orbit. At the depth of about 1.5 cm. the tip of the drill was manipulated so as to widen the area of destruction. In the average dog, this procedure destroyed the entire inner ear. The wound was closed with one mattress suture. As soon as the animals recovered from the anesthesia, the characteristic nystagmus, position of the head and inability to walk verified that the destruction had been accomplished. After a few days the nystagmus

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From the Departments of Otolaryngology and Physiology, Northwestern University Medical School.

1. Ross, E. L., and Fish, M. W.: Ann. Otol., Rhin. & Laryng. **38**:175, 1929.

2. Ross, E. L.; Fish, M. W., and Olsen, A.: Effects of Some Drugs on the Vestibular Response to Rotation, Arch. Otolaryng. **14**:755 (Dec.) 1931.

disappeared and the animals were able to walk normally. About one week after the operation they were used for rotation tests.

To determine the activity of the vestibular system, rotation tests were employed as previously. The animal was placed on an apparatus so that the spinal column was approximately the axis of rotation. This made the plane of rotation vertical to the spine of the animal. This plane of rotation was used because it had been found the best to obtain a nystagmus reaction capable of being increased or decreased, and long enough to be measured with a minimum of error. In making the tests on the animals preliminary to the administration of drugs, interesting results were observed. Tables 1 and 2 contain the data obtained from a number of dogs with one labyrinth destroyed.

The direction of the nystagmus, which has not been tabulated, was opposite to the direction of rotation, as would be expected. However, the striking fact that presented itself was that the reaction was greater when rotation was such that the destroyed labyrinth would ordinarily have been stimulated according to theory. When the right labyrinth was destroyed and the animals were rotated to the right in a vertical plane the duration of nystagmus averaged 8.91 seconds, and when they were rotated to the left the duration of nystagmus averaged 17.77 seconds. Also, when the left labyrinth was destroyed, rotation to the right produced nystagmus lasting 8.99 seconds, while it lasted 18.41 seconds when the animals were turned to the left.

The aforementioned results were obtained by rotating the animal with its spine as the approximate axis. Two positions of the head were used. The animal was attached to a board with the stomach, neck and chin down on the board. The head was turned so that the nose was 45 degrees either to the right or to the left. When the head was turned to the right, the right anterior and left posterior canals were in the plane of rotation. When the head was turned to the left, the left anterior and right posterior canals were in the plane of rotation. When the head was tipped to the right and the whole animal rotated to the right and stopped suddenly, the endolymph pressure was toward the ampulla of the left posterior canal and away from the ampulla of the right anterior canal. When the head was tipped to the left and the animal rotated to the left and stopped suddenly, the endolymph pressure was toward the ampulla of the right posterior canal and away from the ampulla of the left anterior canal. For the sake of making an abbreviated statement, let us assume that positive stimulation is produced by endolymph pressure toward the ampulla and negative stimulation is produced by endolymph pressure away from the ampulla.

The following are the abbreviated statements of the average results given in tables 1 and 2.

TABLE 1.—*Effect on Rotation Tests of Destruction of One Labyrinth* *

Dog	Date	Labyrinth Destroyed	Plane of Rotation	Direction of Rotation	
				Right	Left
20	4/11/32	Right	Vertical	11.8	31.4
		Right	Vertical	12.0	33.8
		Right	Vertical	12.1	29.4
22	4/24/32	Right	Vertical	7.3	26.4
		Right	Vertical	6.9	18.6
		Right	Vertical	6.7	21.8
23	4/25/32	Right	Vertical	0.0	19.6
		Right	Vertical	0.0	18.4
		Right	Vertical	4.0	19.8
19	4/13/32	Right	Vertical	15.5	25.6
		Right	Vertical	12.1	20.4
		Right	Vertical	13.8	16.8
19	5/25/32	Right	Vertical	12.4	13.0
		Right	Vertical	11.0	15.8
		Right	Vertical	8.8	15.7
20	5/29/32	Right	Vertical	8.2	15.8
		Right	Vertical	7.8	16.8
		Right	Vertical	5.8	17.9
21	5/21/32	Right	Vertical	10.4	14.6
		Right	Vertical	13.8	17.2
		Right	Vertical	13.0	14.5
22	5/24/32	Right	Vertical	9.2	15.8
		Right	Vertical	8.3	14.3
		Right	Vertical	8.6	16.6
19	4/30/32	Right	Vertical	8.1	16.1
		Right	Vertical	6.4	15.4
		Right	Vertical	5.6	16.2
20	5/ 9/32	Right	Vertical	7.3	16.8
		Right	Vertical	6.2	15.6
		Right	Vertical	6.0	18.6
21	5/ 4/32	Right	Vertical	10.2	16.8
		Right	Vertical	10.4	17.5
		Right	Vertical	10.2	17.2
22	5/13/32	Right	Vertical	8.5	17.0
		Right	Vertical	8.4	16.0
		Right	Vertical	7.5	19.3
23	5/12/32	Right	Vertical	9.3	14.8
		Right	Vertical	7.8	15.6
		Right	Vertical	9.9	16.5
19	5/27/32	Right	Vertical	10.6	14.2
		Right	Vertical	8.4	14.2
		Right	Vertical	9.8	15.2
20	5/30/32	Right	Vertical	7.4	15.8
		Right	Vertiacl	6.8	16.8
		Right	Vertical	8.3	19.2
21	5/23/32	Right	Vertical	12.0	15.3
		Right	Vertical	11.7	15.8
		Right	Vertical	12.4	16.2
22	5/29/32	Right	Vertical	8.0	13.8
		Right	Vertical	9.2	17.5
		Right	Vertical	8.4	13.2
Average				8.91	17.77

* Results expressed as duration of nystagmus in seconds.

TABLE 2.—*Effect on Rotation Tests of Destruction of One Labyrinth* *

Dog	Date	Labyrinth Destroyed	Plane of Rotation	Direction of Rotation	
				Right	Left
7	2/11/32	Left	Vertical	19.8	10.8
		Left	Vertical	18.1	11.8
		Left	Vertical	20.1	11.2
7	2/25/32	Left	Vertical	14.4	0.0
		Left	Vertical	21.3	0.0
		Left	Vertical	19.0	0.0
9	2/ 4/32	Left	Vertical	19.8	12.1
		Left	Vertical	21.2	7.8
		Left	Vertical	19.0	6.4
10	3/ 4/32	Left	Vertical	39.9	12.0
		Left	Vertical	30.4	1.47
		Left	Vertical	31.8	11.5
4	2/23/32	Left	Vertical	19.9	10.2
		Left	Vertical	22.0	8.9
		Left	Vertical	22.9	10.1
7	2/12/32	Left	Vertical	21.6	16.6
		Left	Vertical	18.2	11.4
		Left	Vertical	17.6	15.2
8	2/27/32	Left	Vertical	13.6	7.7
		Left	Vertical	14.3	8.0
		Left	Vertical	14.0	9.6
9	3/ 5/32	Left	Vertical	14.2	7.9
		Left	Vertical	17.3	6.8
		Left	Vertical	14.8	9.3
10	3/ 5/32	Left	Vertical	26.2	10.3
		Left	Vertical	24.6	8.3
		Left	Vertical	24.8	9.4
4	1/31/32	Left	Vertical	19.5	11.2
		Left	Vertical	17.7	11.2
		Left	Vertical	23.2	10.6
7	2/14/32	Left	Vertical	14.5	7.8
		Left	Vertical	16.4	7.6
		Left	Vertical	15.4	7.6
8	2/29/32	Left	Vertical	12.2	7.8
		Left	Vertical	13.1	7.3
		Left	Vertical	14.4	9.6
9	3/ 9/32	Left	Vertical	13.6	7.6
		Left	Vertical	12.9	
		Left	Vertical	17.2	7.2
10	3/ 9/32	Left	Vertical	16.3	8.1
		Left	Vertical	16.3	10.3
		Left	Vertical	15.8	10.2
4	1/25/32	Left	Vertical	19.1	15.2
		Left	Vertical	18.0	11.8
		Left	Vertical	15.8	13.2
7	2/13/32	Left	Vertical	15.3	7.1
		Left	Vertical	15.9	8.8
		Left	Vertical	16.0	7.7
8	2/28/32	Left	Vertical	13.0	7.1
		Left	Vertical	15.2	8.4
		Left	Vertical	14.8	7.5
9	3/ 7/32	Left	Vertical	18.1	6.0
		Left	Vertical	15.3	6.8
		Left	Vertical	14.4	7.6
10	3/ 7/32	Left	Vertical	21.0	8.0
		Left	Vertical	19.0	8.4
		Left	Vertical	18.8	7.6
Average				18.41	8.99

* Results expressed as duration of nystagmus in seconds.

Right labyrinth gone; head to right; vertical rotation to right: + stimulation of left posterior canal, 9 seconds.

Right labyrinth gone; head to left; vertical rotation to left: — stimulation of right anterior canal, 18 seconds.

Left labyrinth gone; head to right; vertical rotation to right: — stimulation of right anterior canal, 18 seconds.

Left labyrinth gone; head to left; vertical rotation to left: + stimulation of left posterior canal, 9 seconds.

Therefore, "positive stimulation" of the anterior or posterior canals is only about half as strong as "negative stimulation."

Since the foregoing results were uncommon, it was decided to rotate the same animals in a horizontal plane. The animals were attached to a horizontal board on their stomachs and with their noses extended. Such a rotation affected the horizontal canals chiefly. The results of these tests are given in tables 3 and 4.

In measuring the degree of stimulation by the duration of nystagmus, we obtained results opposite to those for the other group of animals. When the right vestibular apparatus was destroyed and the animal was rotated to the right the average animal had a nystagmus lasting 18.72 seconds. With rotation in the opposite direction the nystagmus lasted 10.57 seconds. When the left labyrinth was destroyed and the animal rotated to the right, the nystagmus lasted 10.84 seconds. On rotation to the left, the nystagmus lasted 21.55 seconds.

The following is an abbreviated statement of the average results.

Right labyrinth gone; horizontal rotation to right: + stimulation of horizontal canal, 19 seconds.

Right labyrinth gone; horizontal rotation to left: — stimulation of left horizontal canal, 11 seconds.

Left labyrinth gone; horizontal rotation to right: — stimulation of right horizontal canal, 11 seconds.

Left labyrinth gone; horizontal rotation to left: + stimulation of right horizontal canal, 22 seconds.

Therefore, "positive stimulation" of the horizontal canals is about twice as strong as "negative stimulation."

For convenience, we may speak of three vestibular planes. The horizontal plane is the one passing through both horizontal canals. The "second plane" passes through the right anterior canal and the left posterior canal. The "third plane" passes through the left anterior canal and the right posterior canal. The canals in any of these vestibular planes are so arranged that the open ends of the canals are always forward and the ampullae are always in the more external of the open ends. With sudden stopping after a rotation which has continued for some time in any of the vestibular planes, there must be a tendency for the endolymph to rush toward the ampulla on one side and away from the ampulla on the other. In this paper we are not concerned with questions as to whether there is or is not an actual movement of

endolymph, whether or not the stimulation is through the cilia of the ampullae, or whether the stimulation is due to pressure on the ampullae or some other sensitive spot.

TABLE 3.—*Effect on Rotation Tests of Destruction of One Labyrinth* *

Dog	Date	Labyrinth Destroyed	Plane of Rotation	Direction of Rotation	
				Right	Left
19	5/27/32	Right	Horizontal	17.2	17.5
		Right	Horizontal	14.7	16.2
		Right	Horizontal	16.9	17.8
20	5/30/32	Right	Horizontal	24.0	6.5
		Right	Horizontal	22.8	6.3
		Right	Horizontal	20.1	7.3
21	5/23/32	Right	Horizontal	16.2	9.4
		Right	Horizontal	18.4	9.2
		Right	Horizontal	15.9	10.4
22	5/25/32	Right	Horizontal	16.0	8.4
		Right	Horizontal	13.8	7.1
		Right	Horizontal	12.5	7.1
19	5/25/32	Right	Horizontal	15.3	18.8
		Right	Horizontal	18.6	16.0
		Right	Horizontal	18.4	15.5
20	5/25/32	Right	Horizontal	18.3	8.0
		Right	Horizontal	17.8	9.2
		Right	Horizontal	16.8	8.8
21	5/21/32	Right	Horizontal	17.1	9.2
		Right	Horizontal	12.6	8.6
		Right	Horizontal	16.3	10.0
22	5/24/32	Right	Horizontal	13.7	9.3
		Right	Horizontal	18.7	9.1
		Right	Horizontal	12.4	0.0
19	4/30/32	Right	Horizontal	20.8	21.2
		Right	Horizontal	15.0	17.4
		Right	Horizontal	14.7	16.8
20	5/ 9/32	Right	Horizontal	17.5	8.6
		Right	Horizontal	20.4	9.2
		Right	Horizontal	20.2	9.4
21	5/ 4/32	Right	Horizontal	22.8	11.8
		Right	Horizontal	19.6	10.2
22	5/13/32	Right	Horizontal	16.4	7.4
		Right	Horizontal	19.6	8.2
		Right	Horizontal	18.3	7.8
19	4/19/32	Right	Horizontal	18.6	12.4
		Right	Horizontal	20.3	12.8
		Right	Horizontal	22.4	12.8
21	4/19/32	Right	Horizontal	27.4	11.3
		Right	Horizontal	25.5	9.1
		Right	Horizontal	26.0	11.7
23	4/25/32	Right	Horizontal	23.3	9.9
		Right	Horizontal	27.0	10.4
		Right	Horizontal	23.4	9.4
Average				18.72	10.57

* Results expressed as duration of nystagmus in seconds.

Our results indicate that rotation in any vestibular plane results in a vestibular stimulation which is the sum of the stimulations of both sides and not the difference. If it were the difference, with the one labyrinth gone, a "negative stimulation" of any of the remaining canals

would result in a nystagmus in a direction opposite to the normal. Nystagmus is always in the same direction as it occurs with both labyrinths intact. Therefore, it must be considered that the labyrinth reaction from rotation in any horizontal plane is the sum of the influence of the positive dynamic force on one side and the influence of an equal negative dynamic force on the other side. However, the results clearly prove that the horizontal canals differ from the other pairs of canals. In the horizontal canals the influence of the positive dynamic force is much greater than that of the negative, while in the other canals the reverse is the case.

This same fact was discovered by Ewald [3] through an entirely different method. By means of a "pneumatic piston" he was able to increase and decrease the pressure flow toward the ampulla. When he compressed the ampulla of the horizontal canal, the animal turned its

TABLE 4.—*Effect on Rotation Tests of Destruction of One Labyrinth* *

Dog	Date	Labyrinth Destroyed	Plane of Rotation	Direction of Rotation	
				Right	Left
15	4/ 3/32	Left	Horizontal	8.6	29.2
		Left	Horizontal	9.8	....
		Left	Horizontal	8.7	27.8
		Left	Horizontal	10.8	21.8
		Left	Horizontal	9.8	21.3
		Left	Horizontal	11.0	21.4
16	4/ 2/32	Left	Horizontal	13.1	25.8
		Left	Horizontal	10.2	23.9
		Left	Horizontal	9.0	21.7
16	4/15/32	Left	Horizontal	13.1	14.7
		Left	Horizontal	10.8	15.2
		Left	Horizontal	15.2	14.2
Average				10.84	21.55

* Results expressed as duration of nystagmus in seconds.

head to the opposite side, but when he decompressed the same ampulla the head was turned in the opposite direction but not so forcefully. With the same process in either of the other two canals, the greater reaction followed the decompression. He concluded that in the superior and posterior canals compression brought about an inhibition. We have been unable to find any further work bearing directly on this point. We have also been unable to find any author explaining the difference in the mechanism of the actions of the different canals who does not use Ewald's conclusion.

Since in each labyrinth there is a mechanism to acquaint the animal with movements in any direction in any plane, it might be best to consider that there are two organs of equilibrium and not one. To approxi-

3. Ewald: Physiologischen Untersuchungen über das Endorgan des Nervus Octavus, Munich, J. F. Bergmann, 1892.

mately the same degree as the right eye supplements the left eye and as the right arm supplements the left arm, the right labyrinth supplements the left labyrinth.

The question naturally arises, why should positive pressure toward the ampulla of the horizontal canal be the stronger stimulation and the opposite be the case in the other canals? If a model of the labyrinth of one side is examined, it will be found that the approach to any ampulla is either forward or downward. If the greatest stimulation of all the canals were in the same corresponding direction, the animal with one labyrinth would roll over and over, tending to turn somersaults, owing to the fact that all maximum stimulations were to one side and all minimum to the other. Therefore a difference in the ampullae would lessen the disturbance of equilibrium after the destruction of one labyrinth.

TABLE 5.—*Effect on Rotation Tests of Destruction of One Labyrinth* *

Average				Direction of Rotation	
Number Dogs	Number Tests	Labyrinth Destroyed	Plane of Rotation	Right	Left
5	51	Right	Vertical	8.91	17.77
5	44	Right	Horizontal	18.72	10.57
5	57	Left	Vertical	18.41	8.99
2	12	Left	Horizontal	10.84	21.55

* Results expressed as duration of nystagmus in seconds.

In view of these findings, care must be exercised in the interpretation of the results obtained from rotation tests. If the patient has a dead labyrinth on the right side, a rotation to the left and a quick stop with the head in any position will not result in the absence of nystagmus. Logan Turner [4] recently reported a case with a rather common finding. He said that the patient was "entirely deaf and the caloric reaction was absent" on one side. "The rotation test, however, demonstrated that an equal degree of nystagmus was produced by rotation to the right and by rotation to the left." In our experiments we have always obtained less reaction after rotation and a stop in the horizontal plane, toward the normal side. If rotation were made in any of the other planes, we should expect a greater reaction after rotation toward the normal side.

SUMMARY AND CONCLUSIONS

The labyrinth of one side was destroyed in a number of dogs. Rotation tests were made in all directions.

1. Dogs with but one labyrinth responded to rotation and stop in any plane with nystagmus in the opposite direction to the turning.

4. Turner, Logan: J. Laryng. & Otol. **47**:662, 1932.

2. The duration of nystagmus was approximately twice as long when the animal with one labyrinth was turned in one direction as when it was rotated in the opposite direction.

3. The longer period of nystagmus occurred when the animal was turned in the horizontal plane away from the normal side or when turned in either vertical plane toward the normal side.

4. Facts 1, 2 and 3 lead us to the following conclusions:

A. The tendency of the endolymph to move away from the ampulla in any canal is a stimulation and not a depression or inhibition. It results in the same reaction as if endolymph tended to move toward the ampulla in the canal in the same plane on the opposite side.

B. Each labyrinth is complete in itself, for after the loss of one, the patient may sense movement in any direction.

C. The difference between the responses of the horizontal and other canals to the same action of the endolymph is probably to bring about a neutralization of an unending stream of tonic impulses within each labyrinth. Such an arrangement would result in less disturbance of equilibrium after the loss of one labyrinth than if all ampullae were affected alike.

Clinical Notes

ACUTE INFLAMMATORY SWELLING OF THE SUBMAXILLARY GLAND

Report of a Case Requiring Removal of Both Glands

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Acute inflammatory swelling of the submaxillary gland, characterized by pain and difficulty in swallowing, may be due to many factors. Ivy [1] listed the two most common causes as (1) infection of the tonsils, gums and mouth, causing a submaxillary lymphadenitis, and (2) obstruction of Wharton's duct by calculus or inflammation, causing an acute inflammatory enlargement of the submaxillary gland. Borra [2] mentioned another type of inflammatory swelling of the gland due to bacterial invasion. The specific organism of this type of infection has never been isolated, he stated. Additional conditions affecting the salivary glands, as given by Anthony,[3] include tuberculosis, syphilis, actinomycosis, neoplasms and Mikulicz's disease. Calculi are perhaps the most common cause of symptoms, and their diagnosis and treatment are covered in the writings of Hamlin,[4] Hoover [5] and Harrison.[6]

PHYSIOLOGY

The salivary glands comprising the parotid, submaxillary and sublingual glands have as their function the elaboration and secretion of saliva. According to Howell [7] the most important constituents of this secretion are mucin, a diastatic enzyme known as ptyalin, maltose and various inorganic salts. The digestive action of ptyalin in the mouth is limited to starchy foods, converting starch into maltose and dextrose. The parotid secretion contains no mucin and is richer in ptyalin than the secretion from the submaxillary gland. Another function of the salivary secretion is to moisten the food, and the presence of mucin also acts as a lubricant in the passage of food through the esophagus. Todd and Sanford [8] state that the lower and upper limits of the sugar content of the blood are usually accepted as 90 and 120 mg. per 100 cc., respectively. Hyperglycemia is considered

1. Ivy, R. H.: Swelling of the Submaxillary Region, Ann. Surg. **81**:605 (March) 1925.

2. Borra, E.: Contributo allo studio dei tumori infiammatori della ghiandola sottomascellare, Policlinico (sez. chir.) **35**:345 (July) 1928.

3. Anthony, D. H.: Affections of the Salivary Glands and Their Ducts, J. Tennessee M. A. **24**:175 (May) 1931.

4. Hamlin, F. E.: Calculus of the Submaxillary Gland and Duct, Arch. Otolaryng. **10**:177 (Aug.) 1929.

5. Hoover, W. B.: Obstruction of the Salivary Ducts, S. Clin. North America **11**:303 (April) 1931.

6. Harrison, G. R.: Calculi of the Salivary Glands and Ducts, Surg., Gynec. & Obst. **43**:431 (Oct.) 1926.

7. Howell, W. H.: A Text-Book of Physiology, ed. 4, Philadelphia, W. B. Saunders Company, 1912, p. 740.

8. Todd, J. C., and Sanford, A.: Clinical Diagnosis by Laboratory Methods, ed. 7, Philadelphia, W. B. Saunders Company, 1931, p. 349.

to be present when a value higher than 120 mg. is found. T. and M. Cahane[9] found that the removal of both submaxillary glands in dogs resulted in an increased blood sugar. The animals did not show any signs of cachexia, but the sugar content of the blood of one dog rose from 78 to 201 mg. per hundred cubic centimeters.

REPORT OF CASE

The following case is interesting as it was eventually necessary to remove both submaxillary glands. The right one became diseased first, did not respond to treatment and required removal for the relief of symptoms; in two months the left gland pursued the same course and also eventually required removal.

S. M. P., a woman, aged 23, was admitted to the hospital on June 21, 1931, complaining of pain and swelling in the right submaxillary region, accompanied by difficulty in swallowing, which had been present for the past three days. She stated that the swelling was more pronounced when she partook of food; also that for the past three months the gland would swell at intervals, but would return to normal size in a day. The temperature on admission was 100 F. Examination showed the right submaxillary gland to be slightly swollen and tender. The teeth and gums were in good condition. The tonsils had been removed. Massage of the gland did not produce any flow of saliva from the ampulla of Wharton's duct. Bimanual palpation over the course of the duct did not elicit evidence of any calculi. A roentgenogram gave negative results regarding the presence of a calculus in the duct or gland. The treatment consisted in the use of ice locally, and the patient was placed on a liquid diet. Eight days later the fever and pain had subsided, and on June 29, 1931, the ampulla of the right submaxillary duct was slit for ¼ inch (0.6 cm.) and the flaps were retracted and sutured in position with silk. Passage of a probe failed to meet any obstruction. Treatment thereafter consisted in massage of the gland daily, accompanied by dilatation of the duct and injection of 2 per cent silver nitrate at frequent intervals. Swelling of the gland continued when the patient partook of food and became increasingly disagreeable. It was decided that removal of the gland was necessary for the relief of symptoms, and on Oct. 22, 1931, the right submaxillary gland was removed under general anesthesia. At operation the gland was found to be bound down by many adhesions, and no calculi were found in it after its removal. The pathologic report on the section of tissue was "simple inflammation." Convalescence was uneventful.

In the month of December, two months after the first operation, the patient returned, stating that the same train of symptoms, pain and swelling of the gland on partaking of food, had developed on the left side. Examination again gave negative results regarding the presence of a calculus. The use of ice locally and massage did not give relief, and on Jan. 9, 1932, the ampulla of the left duct was slit and the same treatment as was used on the right side was instituted. The symptoms progressively became more severe, and it was with extreme difficulty that the patient partook of any food, either liquids or solids. On January 29, the left submaxillary gland was removed and was found bound down by many adhesions, as had been the case with the right gland previously removed. The pathologic report was as follows: "The section shows much inflammatory reaction and apparent swelling of the epithelial cells lining the acini. There is no evidence of malignancy or tuberculosis." The convalescence was again uneventful.

The patient's general health since that time has been excellent, and the removal of both submaxillary glands did not appear to cause any untoward results. A

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9. Cahane, T., and Cahane, M.: Hyperglycemia from Removal of Salivary Glands, Compt. rend Soc. de biol. **91**:1232 (Dec. 5) 1924.

determination of the blood sugar made several months later showed 129 mg. per hundred cubic centimeters.

COMMENT

The second submaxillary gland was removed only after all other methods of treatment had failed to give relief and the condition of the patient necessitated this procedure. A search through the American literature and through various textbooks failed to give any information in regard to any untoward results that might be expected from such a procedure. The paper by the Cahanes was found after both glands had been removed.

It would have been more satisfactory if a determination of the blood sugar had been made before and after the removal of each submaxillary gland. However, this was not done, as I was unfamiliar with Cahanes' work at that time. A rise of 9 mg. above the accepted upper limit, while not striking, is suggestive of the fact that the submaxillary glands have an influence on the sugar content of the blood, and that removal of both glands should be performed only after due consideration has been given to this point.

CONCLUSIONS

1. A bacterial infection of the gland may simulate the symptoms produced by calculi in Wharton's duct or in the gland itself.

2. Removal of both submaxillary glands may cause a slight rise in the sugar content of the blood, though the change was by no means striking in the case reported.

A NEW DILATOR FOR THE PHARYNGEAL ORIFICE OF THE EUSTACHIAN TUBE

LOUIS K. PITMAN, M.D., NEW YORK

The isthmus and the tympanic portion of the eustachian tube play no part in the opening and closing movement of the tube. They are constantly open. The pharyngeal portion of the tube is the door to the middle ear. It opens and closes in order to ventilate the middle ear properly, to sweep away any nasal secretion from the orifice of the tube and to break up the surface tension of any secretion stretched across the orifice of the tube.[1] Should the pharyngeal portion fail to open, on account of adhesions, edema or other conditions, the middle ear will suffer.

When one examines the nasal orifice of the eustachian tube with the aid of a nasopharyngoscope, one sees a vertical slit. The tube is closed. During the act of swallowing, or when the patient says "ka," the soft palate rises and there is also visible a concomitant movement of the posterior wall of the pharyngeal orifice of the eustachian tube. This wall is seen to rise, moving inward and backward, and at the same time the mouth of the tube changes its shape from a nearly vertical slit to a triangular opening, formed by a backward movement of the posterior wall of the closed tube. The anterior wall remains stationary. The inferior wall of the open tube is formed by the stretched inferior commissure.

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1. Pitman, Louis K.: Function of the Eustachian Tube, Arch. Otolaryng. **13**:676 (May) 1931.

For a detailed description of the anatomy of the eustachian tube, refer to the work of W. Sohier Bryant.[2]

The present method of dilating the pharyngeal orifice of the eustachian tube consists of passing a direct nasopharyngeal speculum through the mouth and retracting the soft palate forward until the orifice of the eustachian tube is brought into view. The conical end of the dilator is passed through the direct nasopharyngeal speculum into the orifice of the tube. After the instrument has remained there a few minutes, a larger dilator is substituted.

Observation and dilation of the pharyngeal orifice of the eustachian tube are greatly limited when attempted through the mouth, for the following reasons:

1. No matter how much the soft palate is brought forward, the orifice of the tube, on account of its anatomic position, cannot be brought into view in all cases.

2. A great amount of cocainization is necessary, and there is also a great deal of operative and postoperative discomfort to the patient.

3. When the soft palate is retracted forward, its movement is arrested, and the movement of the pharyngeal orifice of the eustachian tube is also arrested, owing

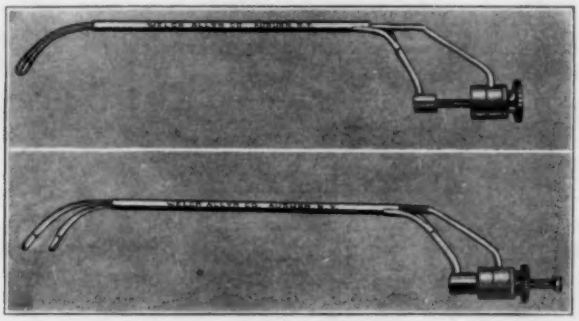


Fig. 1.—The dilator for the pharyngeal orifice of the eustachian tube. The upper view shows the instrument closed. The lower picture shows it fully dilated; this is brought about by the dilating screw at the right.

to the fact that its opening and closing movement works concomitantly with the movement of the soft palate. This method of observation leads one to a partial opinion, for one views the orifice of the tube in an arrested state and hence cannot see whether the tube opens or closes normally.

4. When the soft palate is brought forward in order that one may view the tube, the tube is automatically closed, for the triangular cartilage of the eustachian tube is also brought forward to the anterior wall. When dilation is attempted, two opposing forces are created, a closing force brought about by the speculum and a dilating force brought about by the dilator. Little can be accomplished when two forces work in opposite directions.

In order to overcome these difficulties, I designed a new type of dilator for the pharyngeal orifice of the eustachian tube. The dilator when closed resembles an ordinary eustachian catheter, and it is introduced in a like manner, but under the guidance of a nasopharyngoscope introduced into the nasopharynx through the other nostril. When the dilating portion of the instrument is guided into the

2. Bryant, W. Sohier: The Eustachian Tube, M. Rec. **71**:931 (June 8) 1907.

mouth of the eustachian tube, outside manipulation of the dilating screw will dilate the orifice of the eustachian tube. The instrument is kept in situ for about fifteen minutes. The dilator must always be closed before it is removed from the nose.

In a roomy nose, I use the dilatoscope. This is a nasopharyngoscope which can be adjusted to a dilator carrying a sleeve. Both instruments are introduced into the same nostril. The dilator is inserted first, and the dilating portion is directed toward the orifice of the eustachian tube. The nasopharyngoscope is then introduced through the sleeve of the dilator, and the visual prism is also directed toward the orifice of the eustachian tube. The dilating portion is then directed into the orifice of the eustachian tube, and dilation is effected under direct vision. When the instrument is removed the nasopharyngoscope should be withdrawn first, and the dilator closed before it is removed.

This instrument overcomes all objections to the present method of observation and dilation of the pharyngeal orifice of the eustachian tube through the mouth. It is simple to manipulate, requires little cocainization and can be used in all cases, and only one size is necessary.

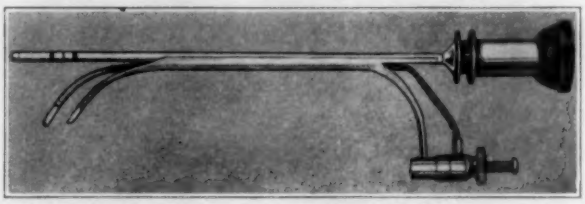


Fig. 2.—The dilatoscope. This is a combined dilator and nasopharyngoscope, both working as one unit. The nasopharyngoscope features the author's shock-proof lighting system.

There is no substitute for the nasopharyngoscope when one is examining the patency of the eustachian tube. It does not arrest the movement of the soft palate, thus giving an impartial picture of the opening and closing movement of the eustachian tube.

This instrument is manufactured by the Welch Allyn Company of Auburn, N. Y., and is sold by all local dealers.

115 West Eighty-Sixth Street.

PAROTID FISTULA FOLLOWING MASTOIDECTOMY TREATED BY ELECTROCOAGULATION

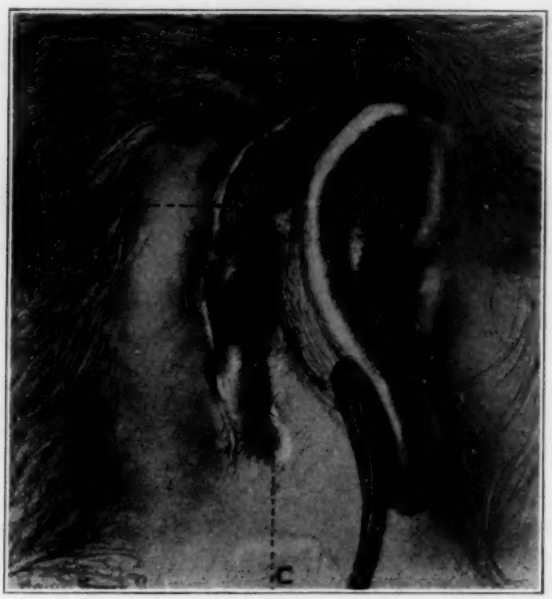
JOHN J. HOCHFILZER, M.D., ST. PAUL

T. P., a girl, now 15 years old, was admitted to the contagious department of the City Hospital eight years prior to the writing of this report. Soon after admission acute otitis media developed in the right ear, which became complicated by acute mastoiditis; mastoidectomy was carried out, and an extensive breaking down of the bone tissue was found. Neither the sinus nor the dura was exposed.

The patient made an uneventful recovery, and when she was discharged six weeks after admission the wound in the mastoid had healed except for a small opening at the lower end of the incision, from which there was a constant seeping of clear, watery fluid.

Seven years later she consulted an otologist in a neighboring city, who concluded that the drainage was due to some diseased mastoid cells which had been left after the operation, and the mastoid was operated on again. The wound apparently healed completely. However, two days after she left the hospital the discharge from the lower end of the first incision began again.

A year after the second operation the patient was seen by me at the Wilder Dispensary; she stated that the only discomfort she had experienced was a constant



A indicates the scar of the first operation; *B*, the scar of the second operation, and *C*, the site of the fistula.

feeling of moisture behind the right ear, and that this moisture increased greatly as soon as she began to masticate, especially tart foods.

Examination revealed the drum membrane fully restored and the hearing normal. At the lower end of the postauricular scar a minute indentation in the skin was noticeable, out of which seeped a clear, watery fluid. An attempt to probe the fistulous tract was unsuccessful. When the patient was given peanuts to masticate, the flow of watery fluid from the wound immediately increased so that I was able to collect 10 cc. in ten minutes. This fluid caused rapid digestion of starch through the successive stages to maltose, indicating the presence of ptyalin, and the conclusion was that this was a salivary secretion.

Numerous treatments have been tried for the closure of parotid fistulas, namely, chemical caustic, galvanocautery, roentgen and radium therapy, excision of the fistula and plastic operations, but none has given great assurance of success. I decided to try electrocoagulation, believing that an agent that was capable

of deep penetration in the smallest possible field offered the best chances of success. With the patient under local anesthesia, a fine needle was inserted for 8 mm. in the direction of the parotid gland, and the current was turned on for ten seconds. This was done several times, the direction of the needle being altered each time in the hope that the fistulary tract might be in the zone of coagulation. During this procedure the facial nerve was watched for any indication of irritation, but none was observed. A few days after this treatment the discharge was considerably lessened, and in ten days it had ceased. Six months have passed since the treatment was instituted, and there has been no recurrence.

It is difficult to draw definite conclusions in regard to the etiology in this case. There appeared to be nothing unusual in the operative procedure. The incision had not been extended unnecessarily downward or forward. It would seem more logical to assume that the parotid gland had extended far backward or that there was an accessory lobe of the parotid.

Lowry Medical Arts Building.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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LARYNGEAL TUBERCULOSIS

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An early diagnosis of laryngeal tuberculosis is so important that a routine examination of the larynx by a competent laryngologist should be made in every case of pulmonary disease.

The early lesions may not produce any subjective symptoms, but in the majority of cases they can be detected by examination with a mirror. Yet there are certain types of lesions that are difficult to differentiate, and there are lesions that appear first in those portions of the larynx that are hidden from the view of the mirror. The most difficult lesion to diagnose is the early infiltrative infection of the ventricle of Morgagni.

Ziegelman [1] called attention to the fact that while prolapse of the ventricle is rare, it is not uncommon to see a simulation of it due to tuberculous infiltration of the ventricle. He even went further and said that bulging of the mucous membrane from the ventricle is almost pathognomonic of a tuberculous process and should be so considered unless proved otherwise.

However, in the early stages of involvement of the ventricle there is no mass projecting from between the true and the false cords, and it is this early lesion that should be recognized as soon as possible.

Recent reported experiences with roentgen examinations of the larynx indicate that another addition to the diagnostic technic has arisen and is deserving of special attention.

Thost,[2] who has been doing this work for a number of years, has become enthusiastic over roentgen examination of the larynx for both the diagnosis and the prognosis of laryngeal tuberculosis. He has examined a large number of normal and diseased larynges, and has said that not only can the early tuberculous lesion be more readily detected

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1. Ziegelman, E. F.: Tuberculoma of the Laryngeal Ventricle Resembling Prolapse of the Mucous Membrane: Report of a Case, Arch. Otolaryng. **15**:382 (March) 1932.

2. Thost, A.: Beitr. z. klin. d. Tuberk. **79**:113, 1932.

by this method than by examination with a mirror, but it is always possible to differentiate between tuberculous and syphilitic lesions. The recognition of the early lesion in the ventricle by roentgenograms is of the utmost value. The normal ventricle appears as a clear air space sharply outlined above the vocal cord, but in the ventricular lesion either this space is entirely obscured or its outlines are much less distinct, with a diminution in the normal clearness of the whole space. Further, if the normal larynx is examined by the fluoroscope during coughing, the air is seen to penetrate into the ventricle, increasing its clearness, but this does not happen if the ventricular mucosa is diseased.

When the tuberculous process begins in the laryngeal mucosa, the cartilages of the larynx soon show variations from the normal. In order to recognize these changes, the examiner must become thoroughly acquainted with the normal larynx. Normally, ossification begins in the larynx shortly after adult life. This is usually clearly made out and is apt to be more marked around the edges of the laryngeal muscles. When tuberculosis is present, decalcification takes place, making the outlines less distinct. The whole picture is less clear and faint.

In contradistinction to this, syphilis can be recognized by the presence of sharp, clearly marked deposits of calcium, especially around the edges of a gumma. It is these contrasting pictures which make a differential diagnosis between tuberculosis and syphilis possible by roentgenograms before it can be made by the laryngeal mirror.

When the epiglottis becomes involved by tuberculosis, its clear linear shape is lost, the shadow becoming almost like a rounded tumor. If it has been destroyed, its absence can be readily noted. When the arytenoids are involved, their sharp lines are lost, and there appears simply a diffuse shadow without clearcut edges. In estimating the clearness of the outline of the larynx in a given picture, it is well to compare it with the sharpness of the outline of the vertebrae.

The importance of roentgenograms in the diagnosis of early tuberculosis of the larynx has also been noted by Wotzilka.[3] He also called attention to the necessity of examining a large number of normal larynges in order to familiarize oneself with the normal picture. In the normal larynx the epiglottis appears as a slender line; the aryepiglottic folds cast slight shadows, and the arytenoid cartilages appear conical. The normal ossification of the cricoid and thyroid cartilages varies in degree with the age and sex of the patient. Ossification is greatest at those points where the muscles are attached, and is more intense and more marked in persons who use the larynx professionally than in others of the same age.

In regard to the ventricle of Morgagni, Wotzilka said that usually it appears as an air-containing space, the lower boundary of which is

3. Wotzilka, G.: Monatschr. f. Ohrenh. **66**:406 (April) 1932.

straight and the upper somewhat curved, but when there is marked ossification of the thyroid, there may be more or less obliteration of this clear area.

In cases of tuberculosis he has noticed certain distinctive changes in the x-ray picture; these changes may be present in varying combinations. When the epiglottis is involved, it has a thick and rounded shape; at times the tip only is involved, and at other times the organ is thickened throughout. When diseased, the aryepiglottic folds throw thicker and heavier shadows, and the arytenoid cartilages appear club-shaped. The thyroid and cricoid cartilages look much smoother and have a less definite shadow.

He also called attention to the fact that syphilis gives a picture absolutely opposite to that of tuberculosis. In cases of syphilis, small sharply circumscribed calcium shadows are seen in both the thyroid and the cricoid cartilage.

As it has been shown by histologic examination that tuberculosis of the larynx frequently begins in the ventricles, probably the greatest aid in the diagnosis of early tuberculosis of the larynx is the appearance of the ventricle of Morgagni in the x-ray film. Changes can be seen here before they can be noted with the laryngeal mirror.

Ross Golden,[4] in his article on the evaluation of roentgenology in otolaryngology, said that ossification of the laryngeal cartilage is a physiologic process beginning before the end of the second decade of life. Although there are variations in its development, the calcification usually begins in the postero-inferior part, involves the lower horns and spreads anteriorly along the lower border. Later, especially in men, it extends to the superior horns and along the upper margin. In the female larynx it is likely to be laid down in dense spots, while in the male larynx it is more diffuse and may have a trabecular structure like cancellous bone.

In spite of other opinions, Golden does not believe that the obliteration of the shadow of the ventricle is of much value as a diagnostic feature, because in half of the normal men studied by Hay the laryngeal ventricle could not be seen, and he himself was unable to demonstrate it in one female larynx. He also believes that destruction of the cartilage may be difficult to detect with certainty.

Tuberculosis, he said, according to Thost and Iglauer, causes decalcification of the cartilages, giving a characteristic washed-out, hazy appearance to the shadow and a thickening of the shadows of the soft tissue in the area involved. On the whole, he is not enthusiastic as to the value of roentgen examination of the larynx as a means to diagnosis

4. Golden, Ross: Laryngoscope **42**:914 (Dec.) 1932.

by itself, but he believes that as a supplement to the mirror and larygoscope of the skilled laryngologist it offers certain possibilities.

Spectorowa, Sobin and Melnikowa [5] expressed the belief that in carefully selected cases of laryngeal tuberculosis in which immunobiologic studies of the patient have been carefully carried out, roentgen therapy, when associated with general clinical treatment, is a worth-while therapeutic measure. In suitable cases favorable results may go on to a clinical cure. The x-rays, however, must be used with caution. They are especially valuable for the small early lesion, and also in cases in which all the pathologic tissue has not been removed by surgical measures. The authors expressed the belief that before the application of the x-rays it is essential to determine the sensitivity of the diseased larynx to ultraviolet rays. This can be done by the external application of the ultraviolet rays in quartz, and if there is no intensive reaction, such as headache, a rise in temperature, hyperemia or swelling of the infiltration, the roentgen treatment can be instituted and the further reactions carefully noted. According to the intensity of the reaction following the application of the x-rays, pauses in the treatment of greater or lesser length are made. The technic of the treatment given by Spectorowa and his associates is as follows: 160 kilovolts and 4 milliamperes of current strength are used, with 0.5 mm. of zinc plus 1 mm. of aluminum filter and a focal distance of 30 cm. The field is covered by a lead localizer 6 cm. in diameter. The larynx is treated on the left and on the right. Each field receives ⅙ unit skin dose, and after from seven to ten days the treatment is repeated.

Grundner,[6] in his article on the therapy for dysphagia, said that as a last resort the x-rays may be used as an aid in the relief of pain. He stated that as a rule he uses only from 5 to 10 per cent unit skin dose with from 2 to 2.5 milliamperes of current strength, 160 kilovolts and 4 mm. of aluminum filter. When he radiates for the relief of dysphagia, from 15 to 20 per cent unit skin dose with the same current and tension is used.

Grundner stated that the greatest pain in laryngeal tuberculosis is caused by involvement of the epiglottis, and that the injection of alcohol into the superior laryngeal nerve produces a certain amount of analgesia in these cases. This relief, however, is limited in duration, though it may last for a few months in some cases. Secondary injections give relief for a much shorter time, and when this relief fails Grundner recommends removal of the epiglottis. His technic for this operation is as follows: Procaine hydrochloride and epinephrine are injected intc both superior laryngeal nerves, and the whole epiglottis and its imme-

5. Spectorowa, Z.; Sobin, J., and Melnikowa, L.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:289 (Dec. 18) 1931.

6. Grundner, G.: Fortschr. d. Therap. **8**:110 (Feb. 25) 1932.

diate neighborhood are painted with a 10 per cent solution of cocaine. Then with a pointed cautery knife the base of the epiglottis on the lingual and laryngeal sides is surrounded with ignipunctures. The epiglottis is removed with Schmidt's forceps immediately above the line outlined by the ignipunctures, and the stump is cauterized with a flat cautery knife. This technic has superseded under his hands the use of the cautery snare, because the snare tends to remove only a small part of the epiglottis, and the rest has to be taken off with forceps. Grundner called attention to the possibility of a spread of the local process after the removal of the epiglottis, and he expressed the belief that there is apt to be an increase in the pulmonary disease.

Instead of making injections into the superior laryngeal nerve for the relief of pain in cases of advanced tuberculosis of the larynx, Nordland [7] advised surgical bisection of the internal branch of the superior laryngeal nerve. He said that the operation has hardly any contraindications, and that it not only relieves sensation, but produces paralysis of the interarytenoid muscles, partially immobilizing the vocal cords. Under procaine hydrochloride infiltration, an incision from 6 to 7 cm. in length is made parallel to the hyoid bone and running midway between the hyoid bone and the upper margin of the thyroid cartilage. It is extended backward across the border of the sternocleidomastoid muscle. The anterior portion of this muscle is retracted, and the position of the jugular vein and the great auricular nerve is noted, and they are avoided. On following the large anterior branch of the superior thyroid artery, one comes to the main trunk of the artery lying on the outer surface of the pharynx, the superior laryngeal nerve being situated a little higher up. Nordland said that he has had no bad results even when the operation has been performed on both sides at the same time.

Nordland's claim that by bisection of the superior laryngeal nerve he obtains paralysis of the interarytenoideus muscle is not in accordance with the established knowledge of laryngeal innervation. The interarytenoideus is innervated in greater part, if not entirely, through the recurrent laryngeal nerve. Exner and others claim that some of the branches of the superior laryngeal nerve are distributed to the lateral crico-arytenoid muscles and the arytenoideus and in some instances to the thyroid-arytenoid muscles, but so far as actual experimentation is concerned, stimulation of the peripheral portion of the superior laryngeal nerve causes contraction only of the cricothyroideus. There is no experimental evidence that any other muscle is excited to action by this procedure.

Muskat [8] also expressed the belief that resection of the superior laryngeal nerve is a justifiable procedure for the relief of pain in cases

7. Nordland, M.: Minnesota Med. **15**:249 (April) 1932.
8. Muskat, I.: Illinois M. J. **62**:57 (July) 1932.

of laryngeal tuberculosis. His preference is to cut the main branch of the superior laryngeal nerve; he said that the accompanying paralysis of the cricothyroideus muscle is not of importance and to dissect out the internal branch requires a good deal more time. The operation causes some motor disturbance of swallowing for a few days, which, of course, is worse after a bilateral resection. Epiglottic pain is not relieved by this operation.

Cemach [9] described a new lamp which is built for the special purpose of getting the ultraviolet rays into the larynx. It is a high pressure mercury vapor arc lamp about 35 mm. long and 10 by 16 mm. broad in oval section. The arc burns in a U-shaped quartz tube, 1 mm. in width. This tube is surrounded by a quartz mantle, and there is a space between the mantle and the lamp through which water circulates for cooling purposes. The mercury vapor arc will burn in any position because of a considerable internal vapor pressure which prevents the thin mercury columns from flowing together when the position of the lamp is changed. The quartz mantle is bent in a circle over its entire length, so that the apparatus resembles a laryngeal instrument. One end is covered with a metal cylinder which is attached to the hand piece; the other end of the quartz mantle, that is, the portion that is to be introduced into the larynx, is covered with a gold sheath which prevents lateral disbursement of the rays. The rays pass out through a window at the terminal end of the lamp, at which point a prism directs the light slightly backward. With the lamp in the proper position, the rays are directed against the ventricular bands and vocal cords from the anterior commissure back to the arytenoids. Usually, other portions of the larynx and even the epiglottis can be irradiated by changing the position of the lamp.

Cemach has also constructed gold covers with side windows which permit lateral irradiation. The lamp is built to run on the direct current, and if an alternating current is used, a converter must be employed instead of a rheostat. The cooling water is supplied to the lamp by two small rubber tubes, one, the intake, attached to the water supply, and the other placed over a bucket so that the operator can hear the splash of the water and thus be sure that there is no cessation of circulation during application of the lamp. Two tongue depressors are furnished with the lamp, one to expose the hypopharynx, and the other with the end so shaped that the epiglottis can be pulled forward. After cocainization of the throat the tongue is forced downward and forward by the depressor, and the lamp introduced. While the lamp in the right hand is being introduced by direct vision, the point is carried behind the epiglottis and down into the larynx until the light almost disappears from the throat. The action of this lamp is intensive, and tissues which

9. Cemach, A. J.: Strahlentherapie **43**:547, 1932.

come in contact with the window will show erythema in from ten to twenty seconds, and vesicles in from twenty to thirty seconds. At a distance of a few millimeters, an application of from forty to fifty seconds will produce erythema. The reddening of the area irradiated lasts for two or three days and should be carefully watched in order to guide further treatment. Treatments should be continued at weekly intervals with increasing doses up to a hundred seconds, and if this dosage is well borne and there is a distinct improvement in the general, as well as the local, condition, the dosage can be gradually increased to two hundred and forty seconds. When there is a marked reaction, the dosage should not be increased and sometimes should be reduced.

Cemach found that ulcerative processes show the greatest improvement after this treatment, and that the lesions with a fibrotic basis also react well. Exudative lesions take a longer time to react, and if there is much surrounding edema, they may be very refractory. Marked infiltration and tuberculomas frequently require months of treatment; this is especially true of the epithelial hypertrophies of the posterior commissure. In this type of case, Cemach uses the cautery, which in the form of ignipunctures is especially applicable for the destruction of tuberculomas. For prolapse of the ventricle he uses a bent cautery knife, the point of which can be placed under the false vocal cord into the ventricle.

It would seem that the Cemach lamp is the best instrument so far devised for getting the ultraviolet rays into the larynx, and its intense action, which requires a very short application, is of great advantage.

Vandevere [10] reported favorable results from the use of the Verba laryngeal mirror, which the patient uses for reflecting sun rays into his own throat. He said, however, that application of the electric cautery is far superior to any other method for treating active tuberculosis of the larynx.

Sir St. Clair Thomson [11] recently published the results of his experience with the use of general irradiation as a therapeutic measure for laryngeal tuberculosis. He employed carbon arc lamps obtained from Copenhagen which are similar to those used in the Finsen Institute. He treated only selected patients, and obtained 75 per cent success. He said, however, that these statistics are misleading, as all of the patients were regarded as favorable as to both local and general conditions, and that some patients had recurrences which healed without treatment with ultraviolet rays. In eleven cases in which there was no improvement, the lesions were healed with the galvanocautery.

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10. Vandevere, W. E.: Southwestern Med. **15**:564 (Dec.) 1931.
11. Thomson, St. Clair: Brit. M. J. **2**:905 (Nov. 19) 1932.

Sir St. Clair Thomson stated in his conclusions that "In thirty-two favourable cases there were no striking evidences of benefit from light treatment; in only two or three might some help from it be claimed. Nor can it be said that healing of laryngeal tuberculosis was hastened, or that the course of light baths in any way rendered subsequent cure by the cautery more rapid or more certain."

The collapse therapy used for pulmonary tuberculosis frequently has a beneficial influence on the laryngeal involvement. Wasowski and Totwen,[12] from observations covering two years of experience with artificial pneumothorax and phrenico-exeresis, expressed the belief that putting the lung at rest favors the healing of the laryngeal lesion by creating a favorable condition for general improvement, and that quieting of the cough and lessening of the sputum distinctly aid in the healing of the local condition. Their experience leads them to believe that the progress of the laryngeal lesion as a rule corresponds closely to that of the lung.

Cooper and Benson [13] treated 106 patients with the collapse therapy and found laryngeal improvement in 40.6 per cent. They also found that the laryngeal improvement was closely associated with the success of the collapse therapy in improving the pulmonary condition, though the larynx did show a slightly higher percentage of improvement than the lung.

In 798 autopsies on patients who had died of pulmonary tuberculosis, Cooper and Benson found demonstrable active tuberculosis of the larynx in 23.3 per cent, and healed laryngeal lesions in about 4.8 per cent. They said that at autopsy it is not uncommon to find laryngeal lesions that show evidence of healing and pulmonary lesions that are progressing.

These results are similar to those that have been obtained previously, and show at least that while one cannot expect much improvement in the local laryngeal lesion if the pulmonary collapse is instituted solely for that reason, the presence of a laryngeal lesion is certainly not a contraindication for collapsing the lung if the pulmonary condition calls for such intervention.

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12. Wasowski, T., and Totwen, S.: Monatschr. f. Ohrenh. **65**:1195 (Oct.) 1931.

13. Cooper, A. T., and Benson, O. O.: Am. Rev. Tuberc. **25**:186 (Feb.) 1932.

Abstracts from Current Literature

Ear

A Case of Partial Deafness Simulating Congenital Auditory Imperception. E. M. Creak, J. Neurol. & Psychopath. **13**:133, 1932.

Creak describes the case of a child, aged 6 years and 10 months, who was neither mentally defective nor apparently deaf, and yet could not utter or comprehend ordinary speech. The child's responses to Head's tests for aphasia were similar to those reported by Worster-Drought and Allen (1929 and 1930) in a case of "congenital auditory imperception." Audiometer tests made by Ewing, however, showed from 50 to 60 per cent loss of hearing for speech. The author concludes that while true congenital auditory imperception probably exists, apparently similar disorders may depend on partial deafness.

McBride, Philadelphia. [Arch. Neurol. & Psychiat.]

Hereditary Disposition to Otitis Media. O. Beselin, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:335 (Sept. 21) 1932.

Beselin presents three genealogical tables to illustrate the importance of hereditary disposition in the occurrence of otitis media. In his opinion, the theories which attribute to the local hereditary constitution of the mucosa a rôle in the pathogenesis of otitis media appear well founded. He thinks that the genealogical tables show the indications for prognosis and therapy of otitis media which a careful family anamnesis may contribute.

Editor's Abstract.

Supernumerary Muscles of the Middle Ear. F. Altmann, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:28, 1932.

In microscopic serial examinations of a large number of malformed temporal bones, Altmann in three instances found supernumerary muscles in the middle ear. He describes the cases in detail because the finding of supernumerary muscles in the middle ear is rare, and because in one case the supernumerary muscles may have been of phylogenetic significance.

Editor's Abstract.

Acoustic Trauma from the Standpoint of the Tonus Theory. K. Wittmaack, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:181, 1932.

The development of his tonus theory has led Wittmaack to new conceptions of the mechanism of acoustic trauma. Experimental production of sound injuries by air conduction in rabbits resulted in morphologic changes analogous to those produced by the experimental application of fluid pressure through the oval fenestra. The author concludes that severe acute acoustic trauma (air-conducted), like contusion trauma, produces an immediate mechanical hydrodynamic lesion not only in Corti's organ but in the nerve fibers and ganglions appertaining to it. Acoustic injuries which are not caused by a sudden severe acoustic trauma but which manifest themselves gradually are produced by a summation of weaker but analogous effects of hydrodynamic pressure. Sound injury produced by bone conduction results in a different morphologic picture; the author thinks that this is due to the fact that the sound conduction to Corti's organ assumes a different form. While in injury produced by air conduction the injurious effect consists in a primary pressure on the labyrinthine fluid which is then transmitted to the endolymph spaces of Corti's organ and to the basilar membrane, in injury by bone conduction the basilar membrane, which is set into vibration directly, together

with Corti's organ, is pressed against the labyrinthine fluid. In the former the destructive process affects primarily the framework of Corti's organ (such as the rods of Corti and arches of Corti), whereas in the latter it affects primarily the cell structures within the framework, especially the sensory cells. Because of the enormous damping effect of the endolymph on the vibrations of the basilar membrane in bone conduction, a sound of given intensity is perceived more feebly in bone conduction than in air conduction, but for this same reason much more severe pressure effects occur within the endolymph spaces of Corti's organ in bone conduction than in air conduction.

EDITOR'S ABSTRACT.

INFLUENCE OF EXTRACTS OF ENDOCRINE GLANDS ON CALORIC EXCITABILITY OF LABYRINTH AND STATIC AND STATIC-KINETIC REFLEXES. T. WĄSOWSKI, Monatschr. f. Ohrenh. **66**:1242 (Oct.) 1932.

On the basis of experiments with guinea-pigs, Wąsowski states that the extracts of endocrine glands exert an influence on the physiology of the vestibular organs which is characteristic for each gland. The extracts of the anterior hypophysis, the thyroid and the thymus stimulate labyrinthine reactions. The extracts of the ovaries and the testicles weaken the labyrinthine reactions. Extensive investigation of the influence of the hypophysis on the labyrinthine reaction showed that only the extract of the anterior hypophysis is able to stimulate these reactions. This stimulation is due to the effect of anterior hypophyseal extract on the sympathetic nerve endings of the ear. The author concludes that the equilibrium of the endocrine glands of the organism favors normal labyrinthine activity. If this equilibrium is disturbed in the direction of overproduction, decrease or disappearance of certain hormones, it must affect the labyrinthine reactions.

EDITOR'S ABSTRACT.

TREATMENT OF OTOSCLEROSIS. HAMM, Monatschr. f. Ohrenh. **66**:1313 (Nov.) 1932.

Hamm has obtained favorable results from the treatment of otosclerosis with high frequency sound waves produced by Mülwert's apparatus. In contrast with some other forms of therapy recently advocated, this treatment is harmless. Treatments are given two or three times weekly for at least three weeks. The results obtained by this method have been so favorable that the author now regards mild otosclerosis as curable, forms of medium severity as susceptible to improvement and only severe forms as incurable; even in these cases the distressing ear sounds often disappear. Several case reports are given.

EDITOR'S ABSTRACT.

STAPEDIUS REFLEX IN MAN. H. KOBRAK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:8 (Sept. 16) 1932.

Kobrak states that the reflex contractions of the tympanic muscles are quantitatively dependent on the exciting acoustic stimulus. In man the observation of the stapedial tendon can be used to determine the true hearing capacity. The author was able to observe this stapedial reflex in the otoscopically normal ear by making the superior posterior quadrant of the tympanic membrane transparent. He describes the technic by which the membrane is rendered transparent. Hearing curves of patients examined in this manner are presented. Clinically Kobrak recommends this observation of the stapedial reflex in cases in which simulation is suspected, to determine whether the degree of sound perception and the degree of contraction of the stapedius correspond. It may also be used in idiopathic dumbness to determine whether the existing hearing capacity is sufficient for the learning of speech. From the physiologic standpoint he is interested in this observation of the stapedius reflex, as it makes it possible to determine the pitch at which maximum contraction occurs in otoscopically normal ears.

EDITOR'S ABSTRACT.

PENETRATION OF IRON-COCAINE SALT SOLUTION FROM THE MIDDLE EAR INTO THE LABYRINTH IN GUINEA-PIGS. F. H. QUIX and A. A. J. VON EGMOND, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:26 (Sept. 16) 1932.

Quix and von Egmond studied the route of penetration of fluids from the middle ear into the labyrinth in guinea-pigs. A mixture of iron salts and cocaine was injected into the middle ear of live guinea-pigs which were killed soon after manifestations of loss of vestibular function (caused by the cocaine) appeared. The skulls were fixed in a solution of formaldehyde and hydrochloric acid, resulting in the immediate precipitation of the iron salts as prussian blue. The author found that in live guinea-pigs the salt solution leaves the middle ear chiefly by the canals of the osseous wall which surrounds the middle ear, flowing directly to the base of the skull, the cranial cavity, the cochlea or the semicircular canals. In the cochlea it penetrates the scala tympani, the scala vestibuli and all the spirals of the cochlear duct. From the scalae it diffuses through the vestibular membrane and the basilar membrane into the cochlear duct. It flows directly around the organ of Corti. It is conducted away from the cochlea by the spiral ligament and flows through the lymph channels along the veins which traverse the osseous wall between two cochlear spirals toward the cochlear axis, finally leaving the labyrinth at the base of the skull. The iron salts cannot be demonstrated in the nerve and the ganglion of the cochlear axis, or in the stria vascularis. They do not penetrate through the fenestra ovalis; occasionally a small amount of salt penetrates through the fenestra rotunda into the scala tympani. In the semicircular canals the salts flow chiefly in the perilymph; they reach the fibrous stroma beneath the sensory epithelium of the cristae and maculae of the utricle and sacculus, so that the sensory epithelium itself is scarcely touched. The ductus and saccus do not take up the salts. The salts do not penetrate through the tympanic opening into the aqueduct of the cochlea; when they have reached the skull they penetrate a short distance into the cerebral opening of the aqueduct of the cochlea, but do not reach the scala tympani. In dead guinea-pigs the salts do not generally flow through the preformed channels, but diffuse slowly through the tissues.

EDITOR'S ABSTRACT.

EXOSTOSIS OF AUDITORY MEATUS AND CHOLESTEATOMA. KRAFT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:79 (Sept.) 1932.

Kraft reports a case of exostosis of the auditory meatus, complicated by a cholesteatoma. The patient, a man, aged 32, consulted him because of suppuration of the left ear of four months' duration and recent attacks of vertigo. The auditory meatus was almost entirely closed by a large exostosis of the posterior wall of the meatus. There was pus in the ear, and pus drained from a fistula about 1 cm. deep behind the left pinna. The hearing capacity was greatly reduced. Surgical removal of the exostosis revealed a large cholesteatoma, which had destroyed the posterior wall of the auditory meatus. During the radical operation, nothing of the auditory ossicles or the tympanic membrane could be seen. The semicircular canals appeared intact. The mastoid process was eburnated. According to the anamnesis, there had been proliferations in the left ear since early childhood, but it was not known whether they were osseous. Baryecoia had existed since the eighteenth or twentieth year.

EDITOR'S ABSTRACT.

INTERRUPTION OF PREGNANCY IN OTOSCLEROSIS. M. GOERKE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:85 (Sept.) 1932.

Goerke discusses the problem of interruption of pregnancy in otosclerosis from a medicolegal standpoint. German law permits the interruption of pregnancy when it is the only method of saving the pregnant woman from an existing danger of death or a severe injury to health. It designates loss of hearing as a severe injury to health. This law brings up the following questions: (1) whether an exacerbation of otosclerosis in pregnancy is to be expected and, if so, under what conditions; (2) whether the exacerbation may be regarded as a severe injury to health accord-

ing to the law, and (3) whether the interruption of pregnancy is the only method of preventing this injury to health. The author thinks that experience has shown that otosclerosis is often exacerbated by pregnancy, but this exacerbation does not occur in every woman with otosclerosis, or with each pregnancy. It is to be expected when the family history reveals frequent cases of otosclerosis and in cases in which an exacerbation is known to have occurred in the first pregnancy. Further, in cases in which there are other calciprivic osteopathies and in which toxic results of pregnancy affect the nervous system, an exacerbation of the otosclerotic process is probable. In a strictly legal sense, otosclerotic disease is not a severe injury to health, as it does not imply total loss of hearing, but the author regards high grade baryecoia equivalent to total deafness in its practical consequences; furthermore, the psychic consequences of the subjective otitic sounds are of equally grave significance and sometimes even lead to suicidal attempts. However, recognition of these dangers does not justify interruption of pregnancy unless it is the only means of averting them. The author has found that the administration of phosphorus and calcium at the beginning of pregnancy prevents the exacerbation of the otosclerosis or reduces it to insignificance.

EDITOR'S ABSTRACT.

BILATERAL MALFORMATIONS OF ORGANS OF HEARING. G. BRÜHL, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:167 (Sept.) 1932.

Brühl thinks that bilateral microtia and atresia are not, as a rule, accompanied by anomalies of the inner ear which cause deafness. Among fifty-three cases of congenital atresia of the ear collected by Marx, only seven were bilateral, whereas the congenital anomalies of the labyrinth which result in deafness are usually bilateral. The auditory vesicle from which the labyrinth develops is early removed so far from the surface that external injuries cannot easily influence its course of development. The superficially located embryonic tissue of the sound-conducting apparatus is more exposed to such mechanical influences as pressure, strain, crushing, adhesion and anomalies of position which are determined by chance and frequently affect only one side. That malformation of the pinna is usually accompanied by defects of the auditory meatus and the middle ear is explained by the common branchiogenous development of all these parts. There is rarely a partial malformation of the sound-receiving apparatus, and it should be especially emphasized that in persons with a normal pinna, malformations seemingly limited to the auditory meatus, such as stricture or atresia, are usually accompanied by malformation of the middle ear. For this reason surgical intervention in cases of microtia and atresia or in those of stricture and atresia of the external auditory meatus for improvement of hearing is usually useless; even if a wide meatus is constructed, the existing anomalies of the middle ear, tympanic membrane, auditory ossicles or tympanic fenestra will prevent a functional recovery. Four case reports are given in support of this opinion.

EDITOR'S ABSTRACT.

ACUTE OTITIS MEDIA AND DIFFUSE SUPPURATING LABYRINTHITIS. T. GERMÁN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:172 (Sept.) 1932.

Germán says that while the diffuse suppurating form of labyrinthitis is anatomicopathologically rather sharply differentiated from the acute labyrinthitis with necrosis of the soft parts of the labyrinth, clinically the rapid progress of the disease process toward the skull is the only distinguishing sign of a necrotic form of inflammation. He reports a case of labyrinthitis with necrosis, in which the suppurative nature of the inflammation could be ascertained only after appearance of the meningeal symptoms. Acute labyrinthine symptoms in the form of subjective disturbances, complete loss of cochlear function and nystagmus of the third degree appeared on the third day of an acute otitis media associated with influenza. While these symptoms indicated a suppurative labyrinthitis, the labyrinthine process was assumed to be of serous nature because labyrinthine

complications arising in the first days or week of otitis media are usually due to serous inflammation. Following antrotomy there appeared to be a quieting of the labyrinthine process, but the continued absence of cochlear function four days after intervention was suggestive, and the appearance of renewed high fever, unilateral headache and nausea with slight bradycardia on the fifth day indicated meningeal involvement. Radical operation with labyrinthectomy was performed. The soft parts of the labyrinth were almost entirely lacking. The temperature became subfebrile the day following operation, and the subjective labyrinthine symptoms as well as the meningeal symptoms disappeared entirely. The author's clinic favors immediate labyrinthectomy in diffuse suppurating labyrinthitis whether it is associated with chronic or with acute otitis media and even if the preparatory antrotomy or attico-antrotomy is not absolutely necessary. Operation on the labyrinth is delayed only if the suppurative character of the labyrinthitis is not positively ascertained, as in the case reported.

EDITOR'S ABSTRACT.

Pharynx

PERITONSILLITIS. R. ELSAESSER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:61, 1932.

Elsaesser says that the typical circumscript peritonsillar phlegmon which does not spread beyond the lateral pharyngeal wall rarely leads to serious complications and has a benign character in spite of its disturbing manifestations and in spite of the conception that most cases of posttonsillitic pyemia are preceded by a spreading of the virus through the peritonsillar tissue. In 160 cases of peritonsillitis observed at the author's clinic there was no serious or septic pyemic complication. The author thinks that the fact that the spread of tonsillar infection usually is by way of the submucosa is favorable for prognosis, as the strong inflammatory reaction produced in this tissue limits the zone of infection and prevents its extension. Why this reaction is sometimes absent and severe sepsis results is not known; the cases reported occurred mainly in healthy, resistant persons. The author found that the more intense the tissue reaction was, the earlier the abscess could be diagnosed, with the result that intervention could be undertaken in time to prevent the suppuration from extending into the parapharyngeal space. The danger of posttonsillitic septicemia is greatest if the abscess shows only slight extension, perhaps having a deep retrotonsillar position, and a reaction of the peritonsillar tissue is entirely or partly lacking, or when the infection does not spread to the peritonsillar tissue but invades the blood stream directly. Under these conditions, the cause of the septic manifestations remains undiscovered until too late for a curative intervention. The majority of cases of posttonsillitic septicemia reported in the literature did not exhibit peritonsillitic symptoms. The author thinks that many cases described in the literature as instances of complications after peritonsillitis may have involved lateropharyngeal abscesses which developed following tonsillitis without intermediary involvement of the peritonsillar region.

EDITOR'S ABSTRACT.

ARTERIES OF THE PALATINE TONSIL. H. BRUNNER and J. SCHNIERER, Monatschr. f. Ohrenh. **66**:1180 (Oct.) 1932.

Brunner and Schnierer studied the arterial supply of the tonsils at necropsy in six specimens and found a much greater variation than was found by Lund in his examination of twenty specimens. They agree with the latter that the arteries usually pierce the superior pharyngeal constrictor at the level of the lower pole of the tonsil and enter the capsule at this level, but contrary to Lund's observations, they also found arteries in the upper pole of the tonsils in cases in which the tonsils were supplied by several arterial branches. In the new-born they found that the arterial branch pierces the constrictor and penetrates from the side through the capsule into the tonsil. The arteries may traverse the parenchyma and ramify on the medial surface of the tonsil, thereby penetrating into the fold of mucous membrane which covers the supratonsillar fossa. In adults they found

the capsule artery, described by Lund, only once, and even in this case there was another artery which appeared under the tonsil and extended to the front to supply the inferior pole of the tonsil and the anterior palatine arch. In the other two cases the arterial branches tended to encircle the tonsil and then penetrate the parenchyma from the front or the back, also supplying, respectively, the anterior or the posterior palatine arch. So far as the derivation of the arterial arteries is concerned they found that in two cases (in a fetus and in a new-born infant) the tonsil was supplied by the ascending palatine artery which originates in the external maxillary artery; in two cases by the ascending palatine and the external carotid, and in one case by two branches of the external maxillary artery as well as the external carotid. The authors agree with Lund that the most important vessels are in the lower pole of the tonsil, and that arterial hemorrhage is most to be feared in this region. They consider it suprising that postoperative arterial hemorrhages are not more frequent under these variable conditions. They attribute the postoperative closing of the tonsillar arteries to their strong annular musculature and the intima cushions of the tonsillar arteries, described by Brunner.

EDITOR'S ABSTRACT.

STRUCTURE OF ARTERIES AND VEINS IN THE TONSILLAR CAPSULE. H. BRUNNER, Monatschr. f. Ohrenh. **66**:1335 (Nov.) 1932.

Brunner discusses the structure of the blood vessels of the tonsillar capsule on the basis of personal examination of many specimens. The arterial vessels of the tonsil are characterized by an unusually thick tunica elastica interna which can be demonstrated in the walls of their finest ramifications. In the larger capsular arteries, intima cushions which do not show a uniform structure are found, frequently in adults and rarely in children. In surgically obtained tonsils one frequently finds artefacts in the larger capsular arteries caused by rupture of the intima and media. The larger capsular veins may be divided into those rich in muscles and those poor in muscles. All these peculiarities in the structure of the capsular vessels are functionally related to the large variations of the blood supply and circulation within the tonsil. The variations in the blood supply of the tonsil are in turn related to the fluctuating occurrence of the secondary nodules and to the growth and involution of the tonsils. Occasionally in the capsule completely thrombosed veins are seen, in the vicinity of which are signs of tissue irritation; in the author's opinion it is doubtful whether or not septic processes originate from these thrombi. Remnants of obturating thrombi are frequently found in large veins in the form of bands of connective tissue traversing the lumen.

EDITOR'S ABSTRACT.

TREATMENT OF BEGINNING DIVERTICULUM OF HYPOPHARYNX. A. SEIFFERT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:256 (Nov.) 1932.

Seiffert thinks that a Zenker's diverticulum in the early stage, which consists in a flat distention of the hypopharynx wall, is not suited to resection and is best treated by division of the musculature of the esophageal opening. This method is safer than a simple stretching of the lumen, and can be executed in a short time and without danger to the mucosa. He used this method successfully in the case of a woman aged 60, who had suffered from painful disturbances of deglutition for two years, and in whom roentgenography showed a greatly retarded passage of the contrast medium through the esophageal opening and a walnut-sized distention of the posterior wall of the hypopharynx. The patient left the hospital nine days after the operation, free from disturbances in deglutition. In later roentgenographic control the distention of the hypopharynx could no longer be seen. This case shows that it is possible to cure disturbances in deglutition caused by hypertonus of the muscles of the esophageal opening by division of the spastic muscles, and also that diverticula are capable of spontaneous regression when the cause of their origin is removed. Up to what degree this regression is possible cannot be concluded without further experience.

EDITOR'S ABSTRACT.

Larynx

A New Treatment for Vocal Disturbances in Unilateral Paralysis of the Recurrent Nerve. E. Fröschels, Monatschr. f. Ohrenh. **66**:1316 (Nov.) 1932.

Fröschels' treatment does not aim at effecting a closing of the glottis but at attaining correct phonation by breath control. The patient is shown that he can attain a good voice without pressing if he avoids excessive breathing. He is then required to do breathing exercises for the purpose of preventing too rapid exhalation. Schreber's home gymnastics are used for this purpose. After a few days the patient practices phonation with breath control, starting with single speech sounds and increasing gradually to syllables, words and sentences. The time required to learn to speak without breathiness, or with a minimum of breathiness, varies with individual ability; but in none of the eleven cases in which this therapy was used did it require more than four weeks to obtain a normal or well sounding speech. Long sentences are spoken in one breath. In all cases the speech was loud enough to be understood at a distance of 10 meters. By effecting the closing of the glottis, other authors hope to bring the paralyzed vocal cord into synchronous vibration with the healthy one to save it from atrophy; the author could discover no sign of atrophy in the only one of his cases which he has been able to observe for three years following the new treatment.

Editor's Abstract.

Therapy of Laryngeal Nodules (Sängerknötchen). L. Stein, Monatschr. f. Ohrenh. **66**:1321 (Nov.) 1932.

Stein thinks that stroboscopic examination is the best guide in the treatment of the round or oval nodules (*Sängerknötchen*) the size of a pinhead that appear on the vocal cords as a result of chronic irritation. Stroboscopy has shown that the mechanism of the vibration of the vocal cords varies with different vocal registers, particularly as to the form of the vocal cords. In examination of a patient with laryngeal nodules the stroboscopic image shows in what form of the vocal cords the nodules hinder adduction inducing hoarseness, diplophonia, fatigue and similar manifestations. It thus shows which form of the vocal cords, that is, which register, should serve as a starting point for vocal exercises. Often it will be seen that even the tones acoustically perceived as correct are incorrectly produced. These are corrected under stroboscopic control, and the corrections are carried over into the speaking voice. The laryngeal symptoms gradually recede, and the nodule itself slowly diminishes. After a time other tones may be sung, in which other forms of the vocal cords are used

Editor's Abstract.

Nose

Diagnosis and Treatment of Sinusitis. Frederick E. Hasty, South. M. J. **25**:1202 (Dec.) 1932.

The author concludes that proper care of infants and children will do much to prevent sinusitis in later life. Care of sinusitis in children consists mainly of general management and nonoperative treatment. Allergy plays an important part in sinusitis in patients of all ages. All possible information regarding the type of pathologic process in a particular case should be at hand before any treatment or surgical intervention is advised. The majority of diseased antrums can be cured by the injection of iodized oil. So-called "piddling" operations usually do little if any good.

Schultz, Chicago. [Am. J. Dis. Child.]

Pathogenesis of Ozena. O. Fleischmann, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:199, 1932.

Fleischmann states that while atrophy of the nasal mucosa, formation of crusts and fetor can be produced by a variety of causes, there is nevertheless a typical

clinical picture genuinely meriting the term ozena, which may be differentiated from disturbances with secondary ozenous manifestations and which has a uniform etiologic basis. He thinks that the origin is best explained by assuming a congenital inhibition of the development of the nasal mucosa. He reviews comprehensively the facts which have been ascertained by anatomicopathologic, clinical, neurologic, bacteriologic, metabolic and hereditary investigations. The fetid and the nonfetid form of atrophic rhinitis represent a gradual variation in the same disease, which develops on the basis of a predisposition inherited according to mendelian laws. This predisposition is purely local; an injury to the organism as a whole acts only as an immediate cause. The bacterial flora of ozena is largely saprophytic. The Wassermann reactions of the blood and spinal fluid are negative. The disease of the mucosa affects the nose and its accessory sinuses and rarely the larynx and trachea; it is characterized by decreased resistance and reaction power. It leads to fibrous degeneration of the mucosa, with the manifestations decreasing as they penetrate deeper, and more pronounced in the main cavity than in the accessory cavities. The osseous changes are in the main independent of the nasal disease process and manifest themselves as inhibitions of development appearing in all parts of the skull but limited to it. The disturbances of the nervous system affect various sensory organs, the sensory tissue of the nose, perhaps also the vagus and the sympathetic, and manifest themselves chiefly in functional deficiency. The author considers the various other theories of the pathogenesis in the light of these facts and finds them all inadequate.

EDITOR'S ABSTRACT.

NASAL POLYPOSIS OF ALLERGIC ORIGIN. C. WIETHE, Monatschr. f. Ohrenh. **66**:1378 (Nov.) 1932.

Wiethe reports a case of nasal polyposis in a worker in a bakery which developed after an allergic rhinopathy which had manifested itself for many years in violent attacks of sneezing and nasal hypersecretion following contact with flour. Repeated surgical interventions were followed by recurrences, but desensitization by intramucosal injections of flour extract resulted in disappearance of the polypi. During early unsuccessful attempts at desensitization it was observed that after a partial desensitization the polypi decreased in size, but grew larger again when the patient was resensitized by particularly severe allergic irritation. This phenomenon was observed several times before complete desensitization was accomplished, and shows that the allergic rhinopathy was the cause of the polypi; they did not disappear during a period of several months in which the patient was removed from contact with allergic irritation. A careful anamnesis in cases of nasal polyposis often reveals the existence of vasomotor disturbances over a period of many years. The author discusses the pathogenesis of polypi based on vasomotor disturbances. He suggests that causal therapy be employed for nasal polyposis when possible.

EDITOR'S ABSTRACT.

HEMORRHAGES AFTER PARACENTESIS OF THE MAXILLARY SINUS. L. MEHLER, Monatschr. f. Ohrenh. **66**:1383 (Nov.) 1932.

Mehler reports six cases in which paracentesis of the maxillary sinus from the inferior nasal meatus was followed by severe hemorrhage. As most hemorrhages originate in the anterior portion of the inferior nasal meatus, or the inferior concha itself, the author advises following Hajek's directions for puncturing the lateral nasal wall at the highest point of the inferior nasal meatus about 3 cm. from the anterior end of the concha. At this point one can, on the one hand, also puncture a small maxillary sinus in the sagittal direction and, on the other hand, avoid the veins in the region of the opening of the nasal duct as well as the arteries of the inferior concha.

EDITOR'S ABSTRACT.

Miscellaneous

CARCINOMA OF THE MOUTH AND TONGUE. JOHN FRASER, Ann. Surg. **96**:481 (Oct.) 1932.

This is an analytic article on the subject indicated, based on a study of sixty-eight cases of carcinoma of the mouth and tongue. Of these, forty-seven were primary in the tongue, all except one involving the anterior two thirds; thirty-two involved the edge, and fourteen, the dorsal surface. The relative immunity of the posterior two thirds of the tongue may be accounted for on the basis of various structural differences in this region, the most important being the larger amount of lymphoid tissue and the relative absence of cellular differentiation in the epithelium. Twenty per cent of all the lesions arose in the palatoglossal region, which may be predisposed by the fact that crypts containing large numbers of epithelial cells which in man are without function, being atavistic remains of highly specialized taste organs in lower forms, may add to the danger of the development of cancer in this region. The floor of the mouth gave rise to 10 per cent of the lesions, the majority of which were grouped about the orifices of the salivary ducts. The factors of age, sex (in the author's figures the ratio is eight males to one female), heredity, syphilis, tobacco, alcohol, mouth infections and direct local irritants and the factor of "epithelial instability" are discussed with commentaries from the author's experience.

The histogenesis of epithelial malignant degeneration is reviewed in accordance with the views generally held by pathologists, and the author lists his cases according to Broders' classification as follows: 48 per cent in group 1, 36 per cent in groups 2 and 3, and 16 per cent in group 4. While the great majority of the tumors were of the squamous cell type, nine atypical cell forms were encountered. Of the sixty-eight patients considered, sixteen had been well two years or longer, thirty-one had had recurrences, twenty had died and one could not be traced. Of the group with glandular involvement (twenty-six cases), only one was well after two years, while fourteen had had recurrences and eleven had died. The author's experience with the use of radium in the treatment of twenty-five patients is briefly reviewed.

WIEDER, Milwaukee. [ARCH. DERMAT. & SYPH.]

EXPERIMENTAL PRODUCTION OF PAROTID TUMORS. M. ABB, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:117, 1932.

Abb attempted to produce tumors of the parotid in nine rats and five guinea-pigs by repeated injection of chemical substances directly into the parotid. The substances used were: scarlet red, scarlet red with balsam of Peru, tar emulsion and diatomaceous earth. The injections were made twice in the first week and thereafter every fourteen days for periods varying between forty-one and one hundred and sixty-six days. Most of the animals also received weekly applications of tar to produce a disposition toward formation of tumors. The injections of scarlet red, scarlet red with balsam of Peru and tar produced changes in the parotid resembling, in large measure, those found by Büngeler and Macchiarulo. Changes in the gland lobules, formation of cysts, metaplastic changes of the excretory ducts with high grade dilatation and, in one case, balls of squamous epithelium and squamous epithelial cysts were seen. The author says that it is a matter of question whether cysts resembling oil cysts can originate from the gland lobules by atrophy of the epithelium. The influence of the experimental disposition to tumor formation is not clearly discernible in the author's experiments; he thinks that this is due to insufficiently long intervals between the injections into the parotid. He attributes to the same cause the deviations from the results obtained by Büngeler.

EDITOR'S ABSTRACT.

CHANGES IN CEREBROSPINAL FLUID IN OTORHINOGENOUS DISEASES. ANNEMARIE NELKEN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:136, 1932.

Nelken discusses the literature and reports the results of her own study of the cerebrospinal fluid. The calcium content of the spinal fluid is constant in normal persons, being between 4 and 5 mg. per hundred cubic centimeters. No decrease of the calcium values in otorhinogenous disease of the brain could be determined. A pronounced increase in the calcium value in the author's observations always indicated meningitis. The calcium value of the withdrawn spinal fluid does not change in the refrigerator or in the incubator at 37 C. during the first twenty-four hours; it changes at a temperature of 56 C. The normal spinal fluid sugar ranges between 50 and 90 mg. per hundred cubic centimeters. Values above 90 mg. are pathologic, but have no specific diagnostic significance. Elevation occurs almost regularly in fractures of the base of the skull. A decreased spinal fluid sugar is pathognomonic for suppurating, but not always for serous, meningitis. The normal and elevated spinal fluid sugar values do not change on standing, but the meningitic spinal fluid sugar value decreases; at 56 degrees the normal, as well as the pathologically altered, spinal fluid sugar content becomes elevated. During encephalography there is no distinct change in the sugar content of the spinal fluid, but there is a change in the blood sugar. The author studied Weichbrodt's reaction and did not find it specific for syphilitic disease of the central nervous system. It is always positive with spinal fluids that have a high cell content, but it can be positive with spinal fluids of normal cell content, in fact in cases in which the Nonne reaction is negative. She thinks that this reaction has no especial diagnostic value in otology.

EDITOR'S ABSTRACT.

PERIESOPHAGEAL ABSCESS. A. HERZOG, Monatschr. f. Ohrenh. **66**:1167 (Oct.) 1932.

Herzog reports a case of a mediastinal gravitation abscess resulting from a manipulative perforation of the esophagus above the level of the clavicle. At this level there was a small triangular abscess cavity to the right of the median line connecting with a narrow elongated cavity extending downward to the left of the median line. Puncture of the upper abscess cavity and drainage resulted in cure. The author points out the similarity between the roentgenologic image of the elongated cavity and that of a Zenker's diverticulum and shows why, in his opinion, it cannot be regarded as such a condition.

EDITOR'S ABSTRACT.

OPERATION ON THE FRONTAL SINUS BY WAY OF THE NASAL VESTIBULE. E. MATIS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:83 (Sept. 16) 1932.

Matis describes an operation on the frontal sinus with an approach through the nasal vestibule. It consists essentially in an incision of the edge of the piriform aperture, extensive detachment of the soft parts and subperiosteal removal of the floor of the frontal sinus and the upper part of the frontal process. The creation of a wide nasal passage is followed by a plastic operation. The author thinks that this method offers all the advantages of the extensive Jansen-Ritter operation, while avoiding facial scars. Therefore he thinks that in all cases in which there are no special complications the endonasal method should be tried first; if difficulties arise, exposure from the exterior can immediately be undertaken.

EDITOR'S ABSTRACT.

PALPATION IN OTORHINOLARYNGOLOGY. H. LEICHER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:199 (Nov.) 1932.

Leicher states that palpation is of great importance in otorhinolaryngology, but its success depends a great deal on the use of the correct method. It is not the tactile sensitivity of the fingertips which is most important in palpation but the motion of the hand. Successful palpation depends primarily on endokinetic

sensitivity (depth sensitivity, muscle sense, motion sensitivity) the receptors of which are in all the tissues of the finger (skin, muscle, tendon, connective tissue, periosteum, etc.). Direct palpation with the finger is comparatively rare in otorhinolaryngology; it may be used for superficial tumors and cysts, and is valuable for the early diagnosis of lingual carcinoma or for locating fishbones lodged in the tonsils or pharynx. Palpation with the fingers through an intervening medium is employed much more frequently. Palpation of the mastoid process is a good example of this method; it is more accurate and reliable than the determination of pressure sensitivity. It is important here, as in all bilateral palpation, that the location, position and tension of the hands be identical, but this must not be accomplished by having the patient turn the head, as that makes the tension of the sternocleidomastoid variable; the only correct position for the physician is directly behind the patient. Every palpation through an intervening medium must be preceded by a topographic orientation. The pressure exerted by the palpating finger should never exceed that which is necessary to come into contact with the object. If the skin is mobile, the underlying parts are palpated by gliding motions in which the skin forms a unit with the finger. This form of palpation is also used for the diagnosis of tumors, emphysema, maxillary cysts and Stensen's duct; exterior palpation permits determination of the form and position of the cartilages of the larynx, their elasticity, mobility, etc. Movement in an object is palpated with the resting finger as in palpation of nystagmus, laryngeal pulsations in aortic aneurysm, etc. Distant palpation, that is, palpation with an instrument, is employed a great deal. It is utilized, often unconsciously, in almost every instrumentation. It plays an important rôle in the introduction of an ear catheter, probing and puncture of the nasal sinuses, anesthesia of the larynx, pharynx and nasal septum and countless other instrumentations.

EDITOR'S ABSTRACT.

Society Transactions

EDITED BY PHILIP E. MELTZER, M.D.

MASSACHUSETTS EYE AND EAR INFIRMARY, OTO-RHINO-LARYNGOLOGICAL SERVICES

Regular Clinical Meetings, Oto-Laryngological Service of F. E. Garland, M.D., and F. L. Bogan, M.D., April 1 to June 30, 1932

OSTEOMYELITIS OF THE FRONTAL SINUS. DR. LEROY A. SCHALL.

This patient, aged 24, was admitted to the hospital with a history of a severe cold in the head for the previous two weeks. The day before admission a slight edema of the right upper eyelid and a severe right frontal headache developed.

Examination showed pus in the right middle meatus, a tender right frontal sinus and a slight swelling of the right upper eyelid.

Roentgenograms showed slightly increased density in the outer angle of the right frontal sinus and bilateral maxillary sinusitis.

The patient was treated conservatively for one week. As the headache became more severe, the edema of the eyelid did not improve and a slight exophthalmos of the right eye appeared, operation was advised.

One week after entrance to the hospital, which was three weeks after the onset, I performed an external frontal (Lynch) and Caldwell-Luc operation. The maxillary antrum and the frontal sinus were filled with thick, creamy pus, the mucous membrane was much thickened, and the ethmoid cells were decalcified. There was no free pus in the orbit. The frontal incision was left open.

The convalescence was uneventful until the thirteenth day after operation. The headache, which had been relieved, reappeared. The temperature, which had been normal, rose to 101 F. A soft, doughy swelling appeared over the right frontal sinus.

DISCUSSION

DR. A. S. MACMILLAN: The first x-ray plates showed slightly increased density of the right frontal sinus at the outer angle. The plates taken thirteen days after the original operation showed definite evidence of osteomyelitis of the right frontal sinus. The outline of the frontal sinus was beginning to disappear.

DR. LEROY A. SCHALL: On the thirteenth day after the original operation, I reoperated by extending the frontal incision across the left side, and by a 4 inch (10.16 cm.) midline vertical incision, the flaps were turned back, exposing the frontal bone. A semicircular line of trephine openings were made, extending from the outer limit of the right frontal sinus to the outer limit of the left frontal sinus and 3½ inches (8.87 cm.) in the midline from the root of the nose. At the uppermost trephine, pus was found in the bone, indicating that I was not beyond the infection. The vertical incision was extended, and a second line of trephine openings was made in the midline 2 inches (5 cm.) beyond the first line. These openings were connected, and the frontal bone was removed, revealing a large extradural abscess. The wound was left wide open, and moist dressings were applied.

DR. H. P. MOSHER: Dr. F. E. Garland and I were present at the operation. When infection was found at the highest trephine, we felt that Dr. Schall had not reached the upper limit of infection of the bone and advised that another set of trephine openings be made higher up on the frontal bone. This was done; the osteomyelitis was completely encircled, and the bone was removed.

LYMPHOMA OF THE NECK. DR. F. E. GARLAND.

This man was sent to us with a diagnosis of tumor of the pharynx. When I first examined him, I thought he had a tumor of the pharynx, but on second consideration I have my doubts. He is 71 years old, a very hearty man, with no complaint except that mucus forms in excess in his throat. If he had a tumor of such a size and it was a cancer, he unquestionably would have much more difficulty in swallowing. He would not be so hale and hearty. The same observations would apply to tuberculosis. You would expect more pain on swallowing or at least referred pain. If it were a sarcoma, he would tend to have bluish veins surrounding the tumor. If the mass were malignant, it would tend to be more adherent rather than several localized masses. I do not believe that the condition is due to syphilis. I am at a loss to make a definite diagnosis. For that reason I removed one of the glands in his neck, and it has been examined by Dr. P. J. Ross.

DISCUSSION

DR. P. J. ROSS: This is a section of the gland. You will note that there is very little stroma, although the outline of the gland and the capsule still remain. There are no germinal centers visible in this gland. A high power view of the cells showed them to be fairly uniform in size, but the nuclei did not stain as darkly as those of mature lymphocytes. All of these cells are immature lymphocytes and classify this tumor as one of the lymphoblastomas. There are a few mitotic figures scattered throughout. There is no abscess formation or evidence of any metastatic growth.

TUMOR OF THE NOSE AND NASOPHARYNX. DR. ANGUS MCLEOD, DR. F. E. GARLAND and DR. LEROY A. SCHALL.

This woman was seen by me (Dr. Garland) with a tumor in the nasopharynx and an infection of the left antrum. The tumor protruded in the nasopharynx as a polypoid mass, and I thought that it was a polyp. It was somewhat rounded and did not seem very hard, and with the history and roentgen evidence of infection of the antrum, I thought that it sprang from the ostium of the antrum and protruded into the nasopharynx. The left side of the nose was completely blocked. The right side of the nose was perfectly clear. I asked Dr. Schall to operate through the canine fossa.

DISCUSSION

DR. LEROY A. SCHALL: At operation we found a chronic infection of the left antrum. There was no evidence of tumor anywhere in the antrum. When the naso-antral wall was cut down, we found a large mass filling the inferior meatus, extending postnasally to the pharynx and the nasopharynx. The inferior turbinate was crowded against the septum, and the tumor seemed to originate from the inferior turbinate directly. It was friable and could not be taken out in one mass. It was completely removed in pieces. The mass involved the entire inferior turbinate.

DR. P. J. ROSS: The first slide shows the lining of the antrum; it is thickened by a deposit of fibrous tissue, but there is very little inflammatory reaction. The second slide is one of the tumor cells, which stained quite uniformly. There is no stroma, which accounts for the extreme friability. Under a higher power lens you see practically the same picture as in the case of lymphoma previously shown, but the nuclei are much larger and there are many more mitotic figures. This section shows a solid mass of immature lymphocytes. From the appearance of this section I should consider it a lymphoblastoma, rapidly growing.

CARCINOMA OF THE TONSIL. DR. F. E. GARLAND.

This man, aged 47, uses tobacco and alcohol to excess. He has had painful, difficult swallowing for from eight to nine months. The throat looks swollen. The tonsil and the base of the tongue are bound together on the left side, and the tongue is pushed toward the median line. There is a large, hard, not very

tender gland in the neck. There is pain referred to the ear. The physical condition is unsatisfactory, and I consider the case inoperable. The patient will be treated with x-rays and radium.

I am showing a second case that I first presented three weeks ago in which I made an attempt to relieve the distressing condition. This man had a carcinoma of the left tonsil and the left lateral wall of the pharynx. The tumor was of several months' duration, was confined fairly well to the left tonsil, the left pharyngeal wall and the anterior pillar and extended into the base of the tongue on that side. It also extended into the lymphatics fairly high in the neck. I could not feel any tumor below this region. I felt justified in the attempt to remove the mass. A blocked dissection of the glands in the neck was done, and at the same time the external carotid was ligated. The patient was allowed to rest for a week, and at the end of that time he was in such a good physical condition that the second stage of the operation was attempted. We began by inserting sutures in the tongue and pulling it out through the mouth so that after prolonged tension on the tongue one might reasonably say that the entire tongue was lifted from its base. This gave an excellent view of the tumor. The extreme tension plus the previous ligation of the carotid made the operation practically bloodless, and that is absolutely essential when working in this manner; otherwise, you spend more time sponging than operating. A radical excision of the mass was attempted, which included the soft palate, right to the ramus of the jaw, along the floor of the mouth, including exenteration of the contents of the floor. The case evidently is an inoperable one, yet I think that the patient's chances of comfort and prolongation of life are better with this procedure than without it. He will also be given high voltage roentgen therapy. The growth was a carcinoma of grade II.

A Thornwaldt Cyst. Dr. Angus McLeod.

This man, aged 30, came to the clinic complaining of deafness in the right ear, which was gradual in onset, but had become much worse the past year. There is also considerable nasal obstruction. Examination showed a large, yellowish mass in the nasopharynx, about the size of a small walnut. On palpation it seemed to be fluctuant. There was retraction of the tympanic membrane of the right ear. There was a yellowish tinge to the membrane. The mass seemed to be attached to the posterosuperior part of the pharynx. X-ray pictures of the cervical spine gave negative observations, and the sinuses were normal.

DISCUSSION

Dr. A. S. MacMillan: From the standpoint of the roentgenologist, the tumor does not have the appearance of ordinary adenoid tissue. It is perfectly smooth and seems to come down from the posterosuperior portion of the pharynx. One cannot say exactly what it is. It may be a simple fibroma.

Dr. G. Tobey: I have seen several of these cysts. The opening becomes blocked, and there is a collection of secretion which becomes infected. I usually decap the whole surface of the mass, and the end-results have been good.

Syphilis of the Esophagus. Dr. P. J. Ross and Dr. H. P. Mosher.

This is a section of the esophagus of one of the specimens Dr. Mosher collected. The patient died of a perforated ulcer of the duodenum. The patient also had syphilis, which was proved at autopsy. This is a section through the lower portion of the esophagus. The rugae are markedly thickened, and the esophagus, as a whole, is considerably thickened. Microscopically, the muscularis mucosa shows tremendous thickening, particularly of the submucosa. This thickening of the esophageal tissues and the diffuse infiltration might be of syphilitic origin. There is definite perivascular infiltration by plasma cells and lymphocytes.

DISCUSSION

Dr. H. P. Mosher: This is the first case of syphilis of the esophagus which I have ever recognized. The whole esophagus showed great thickening microscopically. Otherwise, it looked normal.

DR. D. C. SMYTH: If this is a proved case of syphilis of the esophagus, I think it will be the second case on record. Of a series of 4,000 patients examined by a French investigator, 2 had questionable syphilis, but the diagnosis was not definitely proved in either case.

LARYNGOFISSURE FOR RECURRENT PAPILLOMA OF THE RIGHT VOCAL CORD. DR. F. E. GARLAND and DR. LEROY A. SCHALL.

This patient was operated on in 1930 for papilloma of the right vocal cord. He was well until a month ago, when he returned because of a recurrence of the papilloma and also questionable leukoplakia of a small area of the upper portion of the true vocal cord on the right side. There is no limitation of motion of that cord, and the tumor is growing rapidly. The question now is as to the treatment. The tumor was originally removed by diathermy. Dr. Garland recommends laryngofissure for removal of the tumor.

DISCUSSION

DR. D. C. SMYTH: I believe that laryngofissure should be performed for a recurrence of papilloma in the same site, even if there is no limitation of motion. In this particular case there is beginning keratosis which looks like leukoplakia and is suggestive of a malignant process.

DR. LEROY A. SCHALL: Laryngofissure was performed under avertin and ether anesthesia, and the growth was found on the anterior surface and the anterior third of the right vocal cord. Neither a tracheotomy tube nor a feeding tube was used. Afterward the mucous membrane of the left side above and below the cord, including the entire vocal cord on that side, was dissected and removed. Diathermy was used to control the bleeding after the removal of the membrane. There has been practically no reaction.

DR. P. J. ROSS: This is a section from the vocal cord. It shows the papillomatous growth with a broad base on the surface of the cord, and there is adherent leukoplakia on the surface. It is evidently an irritating lesion, although there is no evidence of malignancy in this section.

LARYNGOFISSURE FOR PAPILLOMA OF THE VOCAL CORD. DR. M. H. LURIE.

This man was first admitted to the hospital two weeks ago for removal of a papilloma at the junction of the anterior and middle third of the left vocal cord. The tumor was removed by Lynch suspension; it was snared off easily. We thought that it was the ordinary type of superficial papilloma. The base appeared clean. A specimen was sent to the laboratory, and the report was that it showed grade 1 carcinoma with some mitotic figures. Laryngofissure was then decided on, and the operation was performed with the patient under avertin anesthesia. A submucous resection was done, the cord being elevated from the thyroid, and the entire cord and mucosa above and below were removed. The bleeding was stopped by diathermy. We did not know whether to sew up the wound tightly or not. I closed the lower part of the wound and inserted one stitch in the upper part without tying it, so that in case of necessity a tracheotomy tube could be easily inserted. There was no complication of any kind. There was some trouble in swallowing for the following two or three days, but since then there has been no difficulty.

DISCUSSION

DR. P. E. MELTZER: Do you think it advisable, Dr. Garland, to use the tracheotomy tube when using diathermy in the larynx?

DR. F. E. GARLAND: Every case should be a law unto itself. I sometimes wonder whether the wounds should be sewed up as much as they are.

DR. D. C. SMYTH: I prefer to insert a small tracheotomy tube for a few days, because if the patients cough, emphysema occurs so rapidly that the tissues blow up before any one can remove the sutures.

DR. P. J. ROSS: This is a section of the papilloma removed; it has a very broad base. In the deeper part of the papilloma there is definite evidence of an infiltrative invasion of the surrounding tissues.

ABSCESS OF THE BRAIN. DR. P. MYSEL.

This patient, aged 27, was admitted to the hospital with the history of having had a simple operation on the mastoid eight years ago. Since that time there had been an intermittent discharge from the middle ear, with headache. On examination, there was a large marginal defect with granulations that protruded through. A radical operation was performed on the mastoid. There was pus under pressure coming from the old mastoid cavity, and exenteration of this cavity exposed a huge extradural abscess. The dura was decompressed for a considerable area. The wound was packed loosely and left open. The patient was returned to bed and observed for the next few days. The only symptom in this interval was a slow pulse, which on admission was 80 and gradually dropped to 50.

During the usual morning visit five days later, the temperature was found to be normal; the pulse rate was 68, and there was no complaint. The patient had no nausea or vomiting. There was no headache. The fundi and fields were normal. There was no evidence of intracranial disease. The same day he suddenly became unconscious, the temperature rose to 105 F., the pulse rate dropped to 56, there was marked stiffness of the neck, and a pronounced Babinski sign was present. Lumbar puncture showed 15,000 cells, with 87 per cent polymorphonuclears. There were no organisms found in a smear. We reopened the wound, and did a further decompression. Exploration of the brain was done with a Cushing needle, and 2 cm. from the surface an abscess was evacuated. I did not permit the pus to exude. Encircling with the diathermy needle I went deeper and deeper until the abscess cavity was entered. Pus immediately exuded through the diathermy wound. I explored the abscess, but did not feel any wall. The depth of the abscess was at least 5 cm. The pus was foul, and from about 30 to 40 cc. was obtained. A large Mosher basket was inserted. For several days following the operation the patient had a stormy time. The temperature remained high, and then gradually decreased. The second lumbar puncture a week afterward showed 10,000 cells, with an initial pressure of 300 cc. The first culture of the spinal fluid was negative. The second culture showed a mixed growth, which was the result of contamination. Examination of the fundi showed nothing exceptional, merely a dilated vein, but no choking of the disk on the right. There was considerable herniation of the brain during convalescence. The Mosher basket was in place for five weeks. It finally extruded itself. At present the herniation is much less. The question now is whether or not to complete the radical operation. Dr. H. Cahill suggested that the radical operation be completed, but that instead of closing over the wound a permanent retro-auricular fistula be left, thereby making for easier treatment. There would be no stroma of tissue over the exposed dura, and it would be a much safer condition for the time being.

TONSILLECTOMY AND ADENOIDECTOMY WITH COMPLICATIONS. DR. W. E. MURPHY.

This child has a history of otitis media and frequent attacks of colds with bronchitis. She is undernourished. The morning after an operation on the tonsils and adenoids there was considerable swelling around the right eye. The upper and lower lids were crepitant to the touch. There is still some emphysema of the lids. Where it come from I do not know. The tonsils and adenoids were removed with the LaForce tonsillectome and adenectome. There was nothing extraordinary about the operation, and no unusual bleeding.

DISCUSSION

DR. D. C. SMYTH: Was there any break in the tissue of the tonsillar fossa, and could you feel any air in the tonsillar region?

DR. W. E. MURPHY: Some air could be felt beneath the ramus of the jaw. There was no air in the tissues of the neck.

DR. A. S. MACMILLAN: A roentgenogram shows definite air in the orbit, around the eyeball, and there is also a pocket of air in the pharyngomaxillary fossa.

DR. W. E. MURPHY: I consulted the ophthalmologists, and they were unable to explain how the air got into the region of the eye.

DR. M. L. HU: I had a patient in China last year on whom I performed local tonsillectomy. The patient struggled a good deal. He had emphysema of the cheeks and over part of the chest. The emphysema subsided in three or four days. In 1929, two cases were reported.

PETROSITIS. DR. C. I. JOHNSON.

A woman, aged 21, 6 feet (182.9 cm.) tall and of normal weight, about 115 pounds (52 Kg.), was seen on March 7 with an acute infection of the ear primarily involving Shrapnell's membrane. She had had headache and earache for two days. On March 25, a simple operation was performed on the left mastoid. The headache persisted, and the temperature was around 100 F. The infection of the mastoid was extensive. It took about three weeks for the middle ear to become dry. At the time of operation, the petrous tip appeared normal in the roentgenogram. The posterior wound healed well after the operation. About four weeks later, slight suppuration of the middle ear began. A roentgenogram showed a few cells in the zygomatic region. These were carefully removed at a second operation. Four or five days after this the middle ear became dry again, but the posterior wound drained for two weeks, and the discharge increased. All this time the headache persisted. The middle ear again began to discharge pus. I inflated the eustachian tube and could force through considerable mucous secretion which I thought came from an infection of the mucous glands in the eustachian tube. I therefore removed the tonsils, which I thought were diseased. Irrigation of the postaural wound with equal parts of hydrogen dioxide and alcohol was begun. A few days later the patient began to get dizzy and presented an irritative type of nystagmus, the quick component being toward the affected ear. The dizziness became worse, and hearing was only fair with the tuning fork lateralized to the affected ear. At this time the nystagmus changed to the opposite ear. A third roentgenogram showed destruction of the petrous pyramid. The roentgenologist, not knowing much about the case, diagnosed the condition as an acoustic neuroma because of the marked destruction about the internal auditory canal. After a careful check-up by complete neurologic examination and examination of the visual fields and fundi, the patient was operated on again, a radical operation being performed on the mastoid. There was no extension of pus from the anterior labyrinthine group of cells, but a definite lead was found through the posterior labyrinthine group into the region of the internal auditory meatus. All the bone between the lateral sinus and the tegmen tympani including these two plates, was removed. An area of blackish purulent granulations was noted deep in Trautman's triangle, and a sequestrum was removed from that region. The progress has been satisfactory since this operation, and the headache has completely disappeared. There is no fever, the dizziness has gone, and the cavity from which the bone was removed is free from granulations.

RESPIRATORY PARALYSIS IN A CASE OF CEREBELLAR ABSCESS COMPLICATED BY HERNIATION OF THE CEREBELLUM INTO THE FORAMEN MAGNUM. DR. G. B. FRED.

A white woman, aged 23, was admitted to the emergency ward of the Massachusetts General Hospital with the history of having had a simple operation on the

mastoid two weeks previously in a neighboring city. Two days after operation, she complained of violent headache and vomiting, and paralysis of the left side of the face was observed. A radical operation was performed on the left mastoid to alleviate this complication, and it was in this condition that she was admitted to the Massachusetts General Hospital.

There was no history of any previous otitic trouble.

At the time of admission, the following signs were noted: stiff neck without a Kernig sign, slight choking of both disks and paralysis of the left side of the face. The tongue was deviated to the right; the reflexes were greater on the right than on the left, and the abdominal reflexes were not present on the left. Lateral nystagmus and ataxia were present on the left side, and past pointing of the left hand was noted. Headaches were paroxysmal, occipital and exceedingly intense. No lumbar puncture was done for fear of forcing the medulla into the foramen magnum.

Because of the patient's maniacal condition, paraldehyde was given to quiet her. When the effects of the drug wore off, she again became wildly maniacal, complaining of terrific headache.

The diagnosis lay between meningitis and cerebellar abscess of the left side.

After consultation with Dr. Bogan, an exploratory operation on the mastoid was decided on in order to settle the question of meningitis, and if this proved not present, to explore the cerebellum for an abscess.

A few minutes after the wound in the mastoid was opened, the patient suddenly stopped breathing and became cyanotic. The heart was regular and forceful. Artificial respiration was instituted, and this brought the patient back to a normal color. However, when artificial respiration was stopped, she again became blue. Attention was immediately directed toward uncovering the cerebellum, artificial respiration meanwhile being continued.

The lateral sinus was uncovered and found to be collapsed. It was packed off, and an effort was made to make an incision through the wall of the sinus into the cerebellum in search of an abscess. However, this was prevented by bleeding. The bone over the cerebellar dura posterior to the sinus was hard and thick, and because time was an important element, a small area of dura anterior to the sinus was exposed and incised, and a Cushing brain needle was inserted. At a depth of 3 cm. in a downward direction toward the base of the skull, an ounce (30 cc.) of thin, green, odorless pus was encountered and a drain inserted. Artificial respiration had been kept up during the entire operation. After thirty minutes, the patient began to breathe spontaneously. Her color came back, and she was put to bed in fair condition.

The organism found by culture was a short chain streptococcus.

Consultation with an ophthalmologist revealed conjugate deviation to the right and 3 diopters of choking in each eye. At times, vertical nystagmus was present.

Nine days later, the patient suddenly showed 5 diopters of choking in the right eye, and 4 in the left, associated with projectile vomiting. A second cerebellar exploratory operation was performed, this time through the wall of the sinus, but no pus was encountered.

Two weeks later, on account of increasing headaches, a third exploration was performed posterior to the sinus; an ounce of thick, green pus was drained. At this time, diathermy was used to remove a cortical cone of cerebellum over the abscess, and a Mosher drain was inserted.

For the next four weeks the abscess drained well. However, toward the end of that time, the patient's pulse gradually became slower until it reached 6 per minute and she died from paralysis of the respiratory center in the medulla. The heart was strong and regular up to the end. Before death a ventricular tap was considered in an effort to release the pressure on the medulla, but her condition would not allow further operation.

At autopsy it was found that the right side of the cerebellum and the medulla had been forcibly jammed into the foramen magnum, producing a herniation or cone of these structures. This accounted for the respiratory difficulty at the original operation and finally was the cause of death. There was no evidence of meningitis.

The large cerebellar abscess was found to have been emptied of pus, showing that the Mosher drain had been in a favorable position for drainage and had done its work well.

Microscopic examination of the cerebellar tissue revealed marked edema.

DISCUSSION

DR. P. E. MELTZER: I think that this case brings up the following point: If respiratory difficulties with obscure findings are encountered during the course of an operation on the mastoid, pressure on the medulla should be considered, and an emergency exploration for cerebellar abscess should be instituted.

TEMPORAL SPHENOIDAL ABSCESS. DR. G. BERNARD FRED.

I was asked to see a 5 year old child in consultation at the Massachusetts General Hospital because he complained of headache and had 3 diopters of choking. There was also a history of discharge from the right ear of one month's duration three months previous to the present consultation. I found that both ears were dry, and there was a healed perforation near Shrapnell's membrane in the right ear. A diagnosis of temporal sphenoidal abscess was made, and admission to the eye and ear infirmary was advised. The physicians in the neurologic department, however, who had also seen the patient in consultation, advised sending him to the neurosurgical service. Dr. Hodgson operated for abscess of the temporal lobe. In making an incision into the right temporosphenoidal lobe, he inserted the needle and felt a mass 2 cm. from the cortex. A needle used for spinal puncture was inserted, and pus was found which on examination was sterile. He then put his finger around the abscess, completely encircling it; he placed a tenaculum on the capsule and evulsed it with the aid of his finger. The capsule was removed in toto. He inserted a drain in the area from which the abscess had been removed. The question was raised as to whether or not to remove the abscess completely or to drain it as we usually do. The wall of the abscess was 3 or 4 mm. thick. The histologic report shows that in the area in which there was infiltration with plasma cells there were accumulations of rounded bodies which were somewhat like yeast organisms, and which, although they resembled yeast organisms, stained more deeply and did not appear to have a capsule as is seen in cases of meningitis due to yeast organisms.

DISCUSSION

DR. D. H. WALKER: I think that as the end-result was unknown it would have been better to have used our method, namely, incision and drainage, the cavity being allowed to fill slowly and to heal from the bottom up gradually.

DR. H. P. MOSHER: This is an interesting case. I know nothing about meningitis due to yeast organisms, and do not remember ever seeing a case here. The manner of treating this patient was dramatic; namely, Dr. Hodgson felt the abscess, found that the pus was sterile, and removed the abscess intact. I cannot imagine anything better than to remove the abscess completely; however, I am inclined to agree with Dr. Walker that it is better to treat these abscesses.

DR. M. MORIARTY: The patient was admitted to the hospital for the first time on Jan. 5, 1932, with acute hemorrhagic nephritis. Two weeks before admission, he had a discharge from the right ear. There was a discharge when he was admitted to the hospital, and drainage was continued for five days. He was discharged on January 20, with the nephritis much improved.

The second admission was on January 24, when the patient had severe convulsions. Roentgenograms of the mastoids taken by Dr. MacMillan were reported as showing both mastoids thickened; the outline of the cells on left side was indistinct. The patient was discharged on February 1, the diagnosis being convulsions of unknown origin, hemorrhagic nephritis and dental caries.

The third admission was on April 12, when there was a history of frontal headaches on the right side and projectile vomiting for from two to three weeks. There were choking of the disks, scarring of the right ear drum and slightly increased knee jerks.

The consultants were: Dr. Solomon of the neurologic service, who thought that the child had an abscess of the brain, probably cerebellar; Dr. Hodgson, who thought that he had an abscess of the brain, probably of the right temporal lobe, and Dr. Fred of the otitic service, who found a healed perforation in Shrapnell's membrane on the right, and who thought that there was probably a temporosphenoidal abscess on the right side.

Dr. Farrell of the ophthalmologic service found 3 diopters of choking in the right eye and 3.5 diopters in the left.

The patient was operated on by Dr. Hodgson on April 16.

Cultures of the pus from the abscess of the brain showed that it was sterile. Cells suggestive of yeast organisms were seen in the removed abscess. Cultures of the spinal fluid were negative until May 5, when anaerobic culture of the fluid obtained on lumbar puncture showed Streptococcus haemolyticus. The pus drained from the right ear and that drained from the wound contained Staphylococcus aureus and the hemolytic streptococcus.

The child's condition became continuously worse, and he had a fluctuating temperature until he died on June 14.

Dr. G. Bernard Fred: At autopsy, a definite meningitis was seen extending over the entire frontal cortex, and pus was found within the ventricles. A flattened, round, cystic mass measuring 3 by 3 by 1.5 cm. was found; the center was occupied by a collapsed cavity which was surrounded by a capsule from 0.3 to 0.4 cm. thick. Some cerebral tissue was attached to the outer surface of the capsule. A number of deeply stained round bodies were noted.

The wall of the capsule contained a considerable amount of dense connective tissue and was thickly infiltrated with plasma cells and a small number of polymorphonuclears. There were several accumulations of polymorphonuclears which had the appearance of small abscesses.

A number of deeply stained round bodies were noted. These varied in size from a little less to a little more than the diameter of a lymphocyte. Microscopically, the bodies may occur singly, but they are usually in groups, and in some cases they appear to be included in large mononuclear cells or possibly in giant cells. They remind one of yeast organisms, but do not appear to have a capsule.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Dec. 5, 1932

Thomas W. Lewis, M.D., *Vice-President*

Effects of Radiation on Allergic Mucous Membrane. Dr. L. B. Bernheimer and Dr. M. Cutler.

This paper will be published in full in the Archives.

Carcinoma of the Epipharynx, with Destruction of the Cranial Nerves, Including Nerves II and VIII. Dr. Howard Ballenger.

A man, aged 40, had carcinoma of the nasopharynx with intracranial involvement. He showed direct extension to, and destruction of, the second, third, seventh and eighth cranial nerves.

A search of the literature failed to reveal a previous case of an authenticated direct involvement of the eighth nerve from carcinoma of the nasopharynx.

DISCUSSION

Dr. Samuel Salinger: This is a complete and excellent report of an unusual condition. I think that most physicians in private practice do not have an opportunity to see many carcinomas of the epipharynx. It is interesting that the diag-

nosis is so frequently missed. I looked up this subject from another point of view, and wish to quote some figures for malignant growths of the epipharynx with particular reference to the results of roentgen and radium therapy. In Bern, seven patients were treated by roentgen rays, and only one lived more than a year. In Prague, eight of ten patients treated by the Coutard method of prolonged irradiation were free from symptoms for a year. A report from Breslau described two cases of carcinoma and seven of sarcoma. No diagnosis was made in three cases. Ten patients were treated with radium only, with no cures; twelve with radium plus roentgen rays, with three cures. From Düsseldorf two cases were reported with no cures. In Erlangen six patients were treated with roentgen rays; four of the cases showed carcinoma, with no cures; two, sarcoma, with one cure.

DR. H. L. POLLOCK: Regarding the cases in which involvement of the eighth nerve occurs with no metastasis, Bruner has just written a splendid article showing the effect of carcinoma on the labyrinth far removed from the ear. I think that a carcinoma of the stomach was found post mortem. There was complete degeneration of the auditory nerve and the labyrinth due to the toxic effect of the tumor, which was far distant from the ear. That was probably the case in the instance that Dr. Ballenger cited, in which there were no metastases but a degenerative effect from the tumor, far removed from the ear.

DR. HOWARD BALLENGER: This patient had a known history of four and a half or five years since the primary glandular involvement. Why he lived so long, and whether his survival was due to the massive doses of roentgen rays or not, I do not know.

I am glad to hear Dr. Pollock's remarks about the degeneration of the eighth nerve, for it shows that one should not draw conclusions in such matters unless an autopsy is performed.

PETROSITIS. DR. HOWARD BALLENGER.

B. F., a youth aged 16, had acute otitis media following influenza. One week later, measles developed. He had persistent elevation of temperature and mastoid tenderness; orbital pain developed about two weeks after the advent of the measles.

A mastoid operation disclosed a very large cellular mastoid. Convalescence was normal for one week; then there was a return of the orbital pain, headaches and fever. The mastoid was reopened and the zygomatic cells were removed. Meningitis developed, followed by death. At autopsy petrositis, a subdural abscess and meningitis were found.

DISCUSSION

DR. JOHN A. CAVANAUGH: This case is especially interesting to me because I recently had a patient who died of this malady. He was convalescing from influenza, and had had a great deal of pain in the left ear for five days. The night before I saw him the ear drum ruptured and discharged. The drum was bulging and had the appearance of a polyp. There was no tenderness over the mastoid or pain on the affected side, but there was pain on the opposite side. I lanced the drum. The discharge was bloody. Cultures were taken which showed the hemolytic streptococcus. The temperature was 102 F. The following day the fever had subsided, the patient felt better, and there was some discharge from the ear, but pain continued in the temple on the opposite side. I suggested that he go to the hospital and have a roentgen examination, but because of economic conditions he refused. The white cell count was 9,400; the red cell count, 4,300,000. The following day he returned, and the temperature was then 100.4 F. The headache on the opposite side continued, and was more severe at night. There was no pain or tenderness in the ear, and very little discharge. There was a good sized opening in the drum. The family doctor was called, but could find nothing definite. Returning the next morning, he found the patient unconscious. He rushed him to the hospital, where the patient died about four hours afterward. A spinal puncture yielded fluid which showed a pneumococcic organism on culture. A postmortem examination was made. The mastoid was opened and appeared

normal. There was no pus or granulation, and there was no area in the middle ear that could be traced macroscopically into the petrous portion, but on laying open the petrous pyramid, large cells filled with pus were found. A culture of the pus in the cells was not made, but from the slide the organism appeared to be a long chain streptococcus. A more detailed report will be given later.

Kopetzky and Almour give one outstanding symptom in petrositis, namely, pain in the ophthalmic division of the fifth nerve. In this case there was no pain on the side in which the ear was involved. They say that these pains are due to inflammation of the dura which produces traction on the ophthalmic nerve and causes pain, but when the perforation takes place, this pain subsides entirely. When there is pain of this type and rupture, why should not pain and the inflammatory condition of the dura continue?

I have dissected this region in six cases so far, and from what I see of the structure around the petrous tip, I think that the symptoms are due to an anatomic variation and depend on the position of the structures.

DR. H. L. POLLOCK: I have been much interested in this work recently, and have dissected a number of temporal bones. I think the opinion is that there are three routes of infection of the petrous. This mastoid was well pneumatized. In cases in which there are sclerotic mastoids there are rarely complications. They occur only in the cases in which pneumatization does not occur. I think that the symptoms can be explained by the route that the infection takes. One route is into the cells above the labyrinth; when this route is followed, the infection breaks through the anterior part of the petrous portion and causes inflammation there, which produces the pain. The second route is a posterior one behind the cochlea; this breaks through into the posterior portion of the petrous bone, and pain does not occur. There is a rapid development of meningitis in these cases. The third route is the sublabyrinthine, along the floor of the antrum, which follows along to the tip and breaks through near the jugular bulb; in these cases there is an abducens paralysis. I have had several cases in which the so-called Gradenigo's syndrome occurred. All these cases clear up following simple mastoidectomy. The infection does not break through, but inflammation is found around the tip.

I have not had much experience with real suppurative petrositis, but I think that when one makes a large mastoid exposure where it is expected, these routes can be followed. In the cases in which the break occurs anteriorly, the inflammation around the gasserian ganglion causes the pain. If the break occurs posteriorly, the patient sometimes has pain. This is true also when the infection breaks through near the jugular bulb.

DR. SAMUEL J. PEARLMAN: I hope that Dr. Wilson will not think it presumptuous of me if I ask his opinion about one point. In the *Acta oto-laryngologica*, a few months ago, an essayist whose name escapes me proposed that the petrous tip be attacked by exposing the anterior vertical canal and going forward between its crura with a fine curet. Is this approach to the petrous tip practical?

DR. J. GORDON WILSON: In discussing petrositis it is well to remember that the cells at the tip of the petrous bone are not comparable to the pneumatic cells in the mastoid. I have examined the petrous bones from over twenty children in the last few years, and in every case I have found that the cells are diploetic cells, marrow cells. Pneumatic cells have been reported in adults, but they must be rare; I have failed to find them in many of my specimens of adult bones when I have looked for them. The diploetic spaces are large and may easily be mistaken for pneumatic cells, especially since the marrow which they contained is often dissolved during the preparation of the bone for sectioning.

In the bones of children who died of otitic meningitis, it is quite common to find an osteomyelitis of the tip. In the suppuration which occurs, the pus, following the line of least resistance, finds an exit in one of two directions, either into the tympanic cavity or under the dura. The prognosis is obviously worse when it forms a subdural abscess than when it opens into the tympanic cavity. Here spontaneous recovery is not impossible. Since the diploetic cells communicate

freely with each other, an early opening anterior to the promontory will often determine the direction and may even tap the suppurating cavity.

DR. HOWARD BALLENGER: I fear that I cannot answer Dr. Cavanaugh's question. Perhaps Dr. Pollock answered it. Sometimes in the Gradenigo syndrome the opposite sixth nerve is involved. Just why, I cannot answer. I think that Lillie's suggestion in regard to the operative technic on the petrous is good. No doubt the normal petrous bone is less cellular than those in which a petrositis is present, and as a result, there is more room for an operative attack from the mastoid in these cellular temporal bones. I think that Lillie's suggestion of entering the petrous bone through the mastoid proper is good, certainly this would be much less dangerous than performing a radical operation as suggested by Kopetzky and Almour. As to Frenckner's report in the *Acta oto-laryngologica* about going in through the superior canal, I do not know whether the results are favorable or not. I have never tried this procedure on a cadaver.

BACTEREMIA AND ABSCESS OF THE BRAIN: ATTEMPTED ORBITAL DRAINAGE OF THE CRANIAL CAVITY. DR. HOWARD BALLENGER.

G. S., a boy aged 7 years, had a sore throat, cervical adenitis, bacteremia, marked orbital edema, paresthesia of the right side of the face and paresis of the left side of the face. Examination of the spinal fluid gave negative results, as did roentgen examination of the sinuses and mastoids. A diagnosis of probable subdural abscess in the region of the sphenoidal fissure, secondary to an orbital infection, was made. Attempted drainage of the subdural abscess through the roof of the orbit was done.

DISCUSSION

DR. WALTER H. THEOBALD: Dr. Ballenger's case is unique and atypical, but at the same time it is pathetic to fail in localizing an abscess of the brain when one feels certain that it is present. The effort seems especially futile, when more than one abscess is discovered at autopsy. Two interesting points are brought out in this report: first, the absence of localizing symptoms; second, the difficulty of locating an abscess of the brain at the time of operation.

Dr. Ballenger's case again reminds us that no part of the human body is capable of harboring disease as carefully and without manifestations as a brain which conceals an abscess. Nor, as Dixon has stated, is there any pathologic lesion that explodes with such sudden and fatal results.

In a large series of necropsies, Körner first established the fact that in more than 85 per cent, abscess of the brain is adjacent to the original focus of infection. Von Bergmann stated that a primary focus is always necessary in the formation of an abscess of the brain. Statistics reveal that over 50 per cent of all abscesses of the brain are otogenous in origin. According to these figures, 35 per cent of the abscesses originate in the sinuses or are the result of trauma; the remaining 15 per cent, therefore, occur as a result of lymphatic or blood stream infection, and a large share of these are mediastinal. These particular lesions come under the management of the aural surgeon less frequently and are far more perplexing in diagnosis than those with an adjacent focus.

On May 18, 1928, I saw a man, aged 46, who complained of severe headache, blurred vision, ringing in both ears, dizziness and nausea. The onset of his illness had been sudden, with chills and fever lasting for two weeks. A diagnosis of malaria had been made and 30 grains (1.94 Gm.) of quinine administered daily. The blurred vision was present for three days. On the date mentioned he was brought to St. Luke's Hospital. He had had recurrent attacks of asthma for many years, but was in fairly good health up to the time of this illness. He appeared uninterested in his surroundings, and his response to questions was slow. He had a desire to get out of bed and wander about aimlessly. He was worried about business difficulties.

Ocular examination showed: vision of 6/200 in both eyes; no diplopia; left homonymous hemianopia for colors; no edema of the disks, and enlargement of the

veins in the right disk. The pulse rate varied from 60 to 72, and the temperature was normal. The hemoglobin content was 83 per cent; the white cell count, 16,500, and the red cell count, 4,160,000. Examination of the spinal fluid gave a count of 600 cells with 33 per cent small lymphocytes and 67 per cent polymorphonuclear cells. No demonstrable organisms were obtained in a four day growth. The results of blood culture were negative. A diagnosis of abscess in the right hemisphere was made, although infection with Torula was suspected. Four days after admission to the hospital the patient died.

Necropsy revealed an abscess of the tracheobronchial lymph nodes, 3 cm. in diameter. At the level of the bifurcation of the trachea this abscess was continuous with the esophagus by a fistulous tract. Another fistulous connection opened into the left bronchus from this abscess. The organisms were Streptococcus haemolyticus, B. coli and S. viridans. The brain contained five abscesses, three in the left and two in the right anterior hemisphere, as large as 5.5 cm. in diameter. There was no abscess in the cerebellum.

It is the opinion of most neurologists that abscess of the brain, secondary to infection of the lungs, is caused by small emboli in the blood stream.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. E. Musgrave Woodman, 22, Newhall St., Birmingham, England.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. J. F. O'Malley, 6, Upper Wimpole St., W. 1, London.
Secretary: Dr. C. Gill Carey, 106, Harley St., W. 1, London.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

Acting President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

SECTIONAL

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary: Dr. W. A. Wagner, 200 Carondelet St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids, Mich.
Secretary: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.
Place: Indianapolis. Time: Dec. 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Walter Bristow, Medical Arts Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Clark, 388 Pearl St., Beaumont, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Dallas. Time: Dec. 1-2, 1933.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. Charles B. Handel, 131 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Carl G. Frost, 37 Allen St., Buffalo.
Place: Buffalo Club. Time: First Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Justin M. Waugh, Cleveland Clinic, Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Tex.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Tex.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Los Angeles County Medical Association, 1925 Wilshire Blvd. Time: 8 p. m., fourth Monday of each month, September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: 6:30 p. m., second Thursday of each month, September through May.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checkers Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. George B. Jobson, 1238 Liberty St., Franklin, Pa.
Secretary-Treasurer: Dr. John B. Simpson, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine, Craig St., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. L. W. Dean, Washington University School of Medicine, St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. C. A. Veasey, Jr., Paulsen Medical and Dental Bldg., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.